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OPHTHALMOLOGY
and
OTORHINOLARYNGOLOGY

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CONRAD BERENS, M.D.
Editor-in-Chief for Ophthalmology

CHEVALIER L. JACKSON, M.D.
Editor-in-Chief for Otorhinolaryngology

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FOREWORD

THE Quarterly Review of Ophthalmology and Allied Sciences and the Quarterly Review of Otorhinolaryngology and Broncho-Esophagology have been combined and will now be published under the title QUARTERLY REVIEW OF OPHTHALMOLOGY AND OTORHINOLARYNGOLOGY. Each issue will include all of the sections heretofore published in the separate journals. Dr. Conrad Berens and Dr. Chevalier L. Jackson will continue as Editors-in-Chief and the Editorial Board will include the entire roster of distinguished authorities who have contributed so much to the progress of the individual Reviews published to date.

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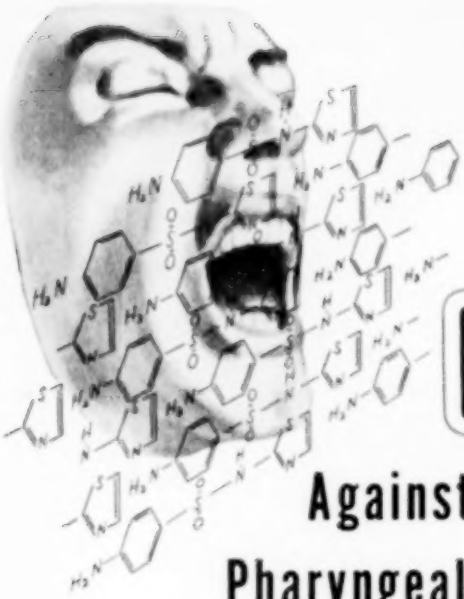

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*Neiman, I. S.: Prophylactic Value of Sulfathiazole, Archives of Otolaryn. 47: 158-166 (Feb.) 1948.

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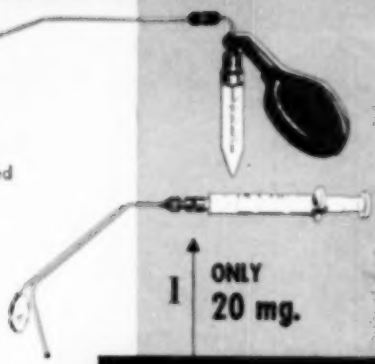

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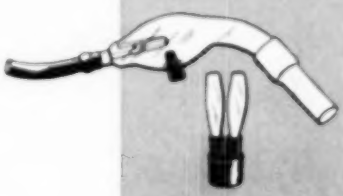

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1. Carabelli, A.: Diseases of the Chest, 15:522, May, 1949 (147 cases; balance to be published).
2. Available from George P. Pilling & Son, Co., 2451 Walnut Street, Philadelphia 4, Pa.
3. Miller, J. B., Mann, F. and Abramson, H. A.: Diseases of the Chest, 16:408, Oct., 1949.

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Quarterly Review
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OPHTHALMOLOGY

Optics, Physiology and Psychology of Vision

Ocular Psychoneuroses. *Edward P. Burch.* Presented at the American Academy of Ophthalmology and Otolaryngology, 1949.

The visual apparatus is richly endowed with potentialities for somatization of psychic conflicts and disturbances of the personality. The structure of the visual apparatus, containing as it does striped and unstriped muscular elements, the lacrimal gland and the retina and optic nerve with their intimate neural connection with the higher centers, make it inevitable that this should be so. Furthermore, as the Freudian psychoanalysts have repeatedly stressed, the eyes possess symbolic value to an exceptional degree.

If one considers the more purely functional ocular neuroses, as opposed to the so-called vegetative neuroses described by Harrington, Zeligs, Schoenberg and others, it seems most convenient and quite feasible to examine them from the standpoint of the conventional terminology of present-day psychiatry. Hysteria, anxiety neurosis, neurasthenia, obsessive-compulsive neurosis and hypochondriasis may and do occur with predominantly visual symptomatology. It would be highly erroneous to assume, however, that these neurotic behavior patterns are hard and fast clinical entities with mutually exclusive symptoms. It should be borne in mind also that the ocular symptoms are often only minor facets of the total neurotic pattern.

For an understanding of their pathogenesis, it is necessary in most instances to trace the development of the personality to early childhood and to make a comprehensive, longitudinal study of the life history, as Adolph Meyer has so convincingly demonstrated. The impacts of early family life, education, sex, marriage, religion, business or profes-

sion all may play an important role as specific, precipitating, intrinsic factors. Extrinsic factors such as wars, economic upheavals, political ideologies, etc. cannot be neglected.

Of the ocular psychoneuroses, ocular hysteria is the most dramatic and has received the most attention in ophthalmic literature. Motivated disregard of visual impressions of any degree up to complete amaurosis, accompanied by corneal and conjunctival anesthesia, and often by bizarre visual field changes as well, is one of the more common ocular manifestations. Motor ties, blepharospasm and photophobia without demonstrable evidence of inflammatory disease are not uncommon in conversion syndromes affecting the eyes. Spasms of accommodation and convergence are other indices of ocular hysteria. Ocular hysteria, as a rule, is best managed by physicians highly trained in psychologic medicine. Those of long standing may require prolonged individual psychotherapy.

Anxiety neurosis is by far the most common type of psychoneurosis seen by the ophthalmologist. The explanations offered by competent psychiatrists concerning the psychodynamics of this type of reaction are numerous. While anxiety is, of course, the etiological constant of all the neuroses, it is a highly predominant feature of anxiety-neurosis. It is important for physicians to realize that anxiety reactions may arise from an iatrogenic background.

Systemic signs and symptoms indicating overactivity of the autonomic system are common. The ocular features usually consist of vague and often bizarre pains in and about the eyes. Sometimes, however, the discomfort is precisely localized. Fear of specific eye diseases or dread of the consequences of overstraining the eyes are not uncommon. Headache is a common symptom. Photophobia may be present and the constant use of tinted lenses may be resorted to without evidence upon examination of redness or congestion of the external ocular tunics. Transient spells of amaurosis, usually described as "blackouts" are common in anxiety-tension neurosis and were especially common in combat personnel during World War II, even persisting after the threat of danger became remote. Marked indecision during the subjective examination for lenses is a common anxiety trait noted in ophthalmic practice.

The neurasthenic patient finds himself unable to use his eyes except for the briefest period of time. Spots before the eyes are often complained of. The patient's power of concentration is usually very noticeably poor.

Obsessive-compulsive states fortunately are not common in ophthalmology. Individuals suffering from this type of mental disorder usually display an unusually rigid type of personality. It has been said that they are punctual, punctilious and parsimonious. Feelings of guilt are not uncommon. They reveal themselves to be preoccupied by

illogical and inconsequential fears and doubts. Ritualistic, repetitive, behavior is the rule in compulsion neurosis. The ophthalmologist may observe patients who are obsessed with the belief that they suffer from glaucoma, cataract, or even strabismus. Meticulous examination and repeated assurances are of little or no avail. In the compulsion types, ritualistic activity involving the spectacles has been observed. Verification compulsions are more common. Obsessive-ruminative-compulsion neuroses demand expert psychiatric therapy and are notoriously refractory to even prolonged and intensive psychotherapy.

True hypochondriasis is not common in ophthalmology. Excessive pre-occupation with the gastro-intestinal tract is almost a universal complaint. Many hypochondriacs wander from refractionist to refractionist demanding a change of glasses. Seldom, if ever, is it possible to completely satisfy the patient that his lenses are correct. It seems useless for the ophthalmologist to attempt treatment of the hypochondriac.

As a rule, ophthalmologists should restrict their psychotherapeutic endeavors to the anxiety-tension and neurasthenic groups of patients. Treatment at the ophthalmological level is also best confined to psychoneuroses of recent origin and those which are definitely topical. Those of iatrogenic and semantogenic origin are particularly susceptible to improvement by the ophthalmologist if he is willing to devote sufficient time to the project and has no false conceptions concerning the fundamental nature of psychosomatic illness. Placebos are valueless and often do more harm than good. Minor changes in correcting lenses and the correction of minor refractive errors for the most part must be classified as placebos, and if they apparently benefit the patient they probably do so because of suggestion or transference (in its widest sense) rather than because minor errors of refraction or phorias of low degree produce disabling symptoms in emotionally well-balanced individuals.

It is sometimes possible to assist the psychoneurotic patient to gain insight and recognize and acknowledge the fundamentally psychic basis for his complaint and symptoms by prescribing a reading course of books which explain the mind-body relationship. These must be supplemented by occasional interviews with the ophthalmologist.

Finally, it should be said that since it is impracticable for most obvious reasons to refer all psychosomatic eye problems to the psychiatrist, a thorough knowledge of psychosomatic principles is highly important to the ophthalmologist today.

Aniseikonia for Distance and Near Vision. *Kenneth N. Ogle and Wendell Tretter.* Am. J. Ophth., 32(12): 1713–24, Dec. 1949.

The design of the eikonometer necessitated investigation to deter-

mine whether there were any differences between aniseikonic measurements for near and those for distance.

The data on 115 patients examined on the standard (direct-comparison) eikonometer and on the space-eikonometer at the Dartmouth Eye Institute (before its closing in 1947) are reported. Both techniques are briefly described.

Study of the available data revealed that, within the precision with which the measurements can be made, the amount of aniseikonia measured for near vision is essentially the same as that measured for distance vision.

Diagnostic Methods of Examination, Biomicroscopy and Photography

Cross Cylinder and Dial Tests for Astigmatism. *William H. Crisp, Denver, Colo.* Presented at the American Academy of Ophthalmology and Otolaryngology, 1949.

Although the retinoscope and ophthalmometer have distinct value, subjective measurement of refraction is usually practicable, more precise, and more reliable than objective testing. But subjective testing should be given as nearly as possible an objective quality. It does not yield best results unless the examiner is constantly alert to discover the full measure of the refractive error. In an important percentage of cases, maximum accuracy is not likely to be attained without the use of a cycloplegic. Much has been said about attaining accuracy within a margin of error of a half diopter, but this is not an adequate degree of accuracy. It is also grossly improper to aim at a supposed general standard of visual acuity rather than the optimum correction and the optimum visual acuity for the individual who is being tested.

By applying the cross cylinder with the Crisp test (Annals Ophth. and Otol. 26: 42, 1917) for refinement of measurement of amount of cylinder and the Stine test (Crisp and Stine, A. M. A. Section on Ophthalmology, 1948) to refine testing for axis, it is possible in most patients to measure the astigmatic error very exactly without dependence upon the letter chart except as to the spherical error.

With the letter chart the cross cylinder test as to amount (after approximation of the sphere by the fogging method) affords the patient essentially a choice between overstatement and understatement of the amount of cylinder required; and the cross cylinder test for axis affords the patient essentially a choice between displacement of axis of the trial cylinder in two opposite directions. Using the rotating astigmatic dial with the cross cylinder, the patient is offered similar choices as to the test for amount and the test for axis. But approximate estimation of sphere by the fogging method preferably precedes all these uses of the cross cylinder.

Earnest pursuit of complete analysis of the patient's refraction is necessary for the best results. Speed in refraction work is by no means always desirable. Attempts to estimate mathematically the exact spherocylindrical equivalent produced at each stage of cross cylinder testing are apt to puzzle and disconcert the refractionist unnecessarily and without benefit.

Some patients cannot be satisfactorily tested with the astigmatic dials, while others cannot be satisfactorily tested with the cross cylinder, at least as far as strength of cylinder is concerned. It is well to employ both methods, one checking the other when necessary. This is particularly advisable in estimating amount of astigmatism in vertical and horizontal positions, for in these positions the cross cylinder test for amount of astigmatism is sometimes misleading.—*Author's abstract.*

Ocular Movements and Motor Anomalies, Nystagmus, Reading Disability

Congenital Anomalies. *Rudolf Aebli, New York, N. Y.* Presented at the American Academy of Ophthalmology and Otolaryngology, October, 1949.

Congenital Anomalies present a varied and complex picture, and it is of the utmost importance to remember that the surgical treatment, if any, must frequently depend on the findings at operation. Anomalies of this type may be due to abnormal fascial ligaments and bands, to structural muscle defects, to abnormal muscle insertions, or to aberrant and abnormal neurological development. The more usual congenital anomalies were dealt with, giving points of diagnosis and differential findings.

Retraction Syndrome: This group of cases is characterized by all or some of the following peculiarities. 1) Complete, or less often, partial absence of abduction in the affected eye. 2) Partial, rarely complete, deficiency of adduction in the affected eye. 3) Retraction of the globe in adduction, rarely in abduction. 4) Narrowing of palpebral fissure in adduction, widening of fissure in abduction. 5) An oblique movement of affected eye either up and in or down and in when impulse to adduct is exercised. 6) Remote near point of convergence.

Strabismus Fixus: This is a congenital condition in which the eye is anchored in the nasal field and in which one attempted abduction the eye does not come out to the midline. It is due to a broad short fibrous rudimentary band, intimately attached to the sclera near the equator.

Marcus Gunn Syndrome: This condition is characterized by a more or less marked ptosis with alternate elevation and depression of the lid on mastication. In contra-lateral jaw movements the lid is excessively elevated, in ipsilateral movements the lid droops and the fissure closes.

A weakness of both elevators in the affected eye may be present. It is due to a neurological anomaly, involving aberrant connections between the motor branch of the 5th with the 3rd nerve nucleus.

Paralysis of Both Elevators of an Eye: This may be bilateral but is most frequently unilateral. Ptosis may or may not be present. The inferior rectus is not involved except in secondary contracture. The ptosis may be real or only apparent as the lid follows the hypotropic eye down. Either the paretic or the non-paretic eye may be used to fix. Binocular single vision may be obtained in the lower field. The importance of these factors in the surgical treatment was discussed. Correction of the vertical deviation should precede any correction of the ptosis.

Congenital paralyses of the superior rectus, the inferior and superior obliques, and of the inferior rectus were discussed.—*Author's abstract.*

The Vertical Deviations. *Ernest A. W. Sheppard, Washington, D. C.* Presented at the American Academy of Ophthalmology and Otolaryngology, October, 1949.

Vertical deviations were discussed under the headings given below.

I. Comitant.

A. Right hypertropia.

1. Monocular: Compare with comitant right esotropia.

a. Left eye fixing.

The right eye deviates up. Cover the left eye—both eyes rotate down, the right to fix, the left to deviate down, *i.e.*, left hypertropia. Uncover the left eye—both eyes rotate up, the left to resume fixation and the right eye to resume deviating up. The degree of deviation does not vary materially with the fixing eye, the tipping of the head or the cardinal positions of gaze. Diplopia test—right image lower. Positive vertical divergence (measured with prisms base down in front of right eye) increased, negative vertical divergence decreased. Rotations normal.

b. Right eye fixing.

Findings similar except that the left eye deviates down, *i.e.*, comitant left hypotropia.

2. Alternating: Compare with alternating comitant esotropia. Either eye fixates. When the left eye fixates the findings are similar to (b) above.

Treatment: If under 10 Δ, prism base down right and base up left. If higher than 10 Δ, advance the inferior rectus or recess the superior rectus of the right eye, if it is the deviating eye. In older patients if without symptoms, masterly non-interference.

B. Left hypertropia is opposite to right hypertropia.

II. Non-Comitant.

A. Paralytic.

1. Infranuclear.

a. Single muscle involvement, *e.g.*, right superior oblique paralysis. Compare with right external rectus paralysis.

i. Recent stage. Diplopia, right image below. Greatest separation of the images in looking down and to the left, the field of action of the paralyzed muscle, and more so with the red glass in front of the left eye (secondary deviation) than with it in front of the right eye (primary deviation). Limitation of motion if the paralysis is complete. Right hypertropia increases with the head tipped to the right shoulder, decreases or disappears with the head tipped to the left shoulder. Right eye fixing = left eye deviates down; left eye fixing = right eye deviates up.

ii. Intermediate and late stages. Three conditions may follow, after a varying length of time, the recent onset of paralysis.

x. Enough of the characteristics of the paralysis may remain so that diagnosis can be made, although not as easily as at the onset.

y. A gradual change from a noncomitant to a comitant strabismus takes place. Thus the right hypertropia may be about the same in the right lower field as in the left lower field, or the deviation may be the same in the left upper field as in the left lower field. That is, the change to comitance may occur either in the lower or the left fields first and later in the right and upper fields. The field of least deviation may be diagnostically significant. There is now no field in which the deviation is definitely greatest.

z. Difficult and confusing are those cases in which there has been practically complete recovery of the paralysis but in which during the recovery period changes have taken place in the direct antagonist (right inferior oblique), or in the contralateral antagonist (the left superior rectus). Secondary contracture or spasm, so-called, of the right inferior oblique may result in the greatest deviation being in the upper left field—not in the lower right field as in the recent stage. Hence, in the late stage it cannot be stated dogmatically that the field of greatest deviation is the field of action of a paralyzed muscle. "Inhibitional palsy" (Chavasse) of the left superior rectus (under-action according to Hering's law—Adler) may simulate paralysis of this muscle.

What changes occur depends on the etiology, the completeness of the paralysis, how long it lasts, the amount of recovery, the fixing eye and the fusion status.

Comparison of paralysis of right superior oblique and of left superior rectus:

MUSCLE	TORS	ANTAGONIST	UNDERACTION	PTOSIS	HEAD TILTING
R.S.O.	L.I.R.	R.I.O.	L.S.R.	Pseudo	R. Shoulder RH inc L. Shoulder RH dec
L.S.R.	R.I.O.	L.I.R.	R.S.O.	True	Not characteristic

Treatment

Recent: Etiology. Occlusion of eye with the paralyzed muscle.

Intermediate: Continue occlusion or prisms according to deviation in lower field.

Late: Surgery, if symptoms.

b. Multiple muscle involvement. More than one muscle in one eye or one or more muscles of both eyes. Motion picture of paralysis of the right superior oblique and of the left inferior oblique, and of both elevators of one eye. Belly phenomenon.

2. Nuclear, internuclear and supranuclear. Bielschowsky. Walsh.

B. Pseudoparetic.

There is no vertical deviation in the primary position. As the eyes are rotated to the left, the right eye deviates up when fusion is interrupted by the nose. As the eyes are rotated to the right, the left eye deviates up when fusion is interrupted by the nose. It may be unilateral or bilateral. No paralysis of the homolateral superior oblique or the contralateral superior rectus.

Treatment: Surgery contraindicated. Problem purely cosmetic and psychologic. Avoid looking far to the right or to the left.

C. Dissociated.

Cover or place a dark glass in front of the right eye and it slowly "wanders" up. Remove the cover or dark glass and the eye slowly "wanders" down to the horizontal. Similarly the left eye. This wandering is present in all positions of gaze. Maddox rod or red glass in front of the right eye—the right image is below. Maddox rod or red glass in front of the left eye—the left image is below. The vertical divergences, positive and negative, are greater than 10 Δ.

Treatment: Prisms and surgery contraindicated. Orthoptic training directed to fusion. Often there is a psychologic or neuropathic factor. Constitutional measures as indicated.

D. Combinations of the above.

Atypical cases which cannot be classified.—*Author's abstract.*

The Management of Strabismus. *Edmond I. Cooper, Detroit, Michigan.*

Presented at the American Academy of Ophthalmology and Otolaryngology, 1949.

The management of strabismus includes accurate diagnosis, refraction and glasses, treatment of amblyopia, pre-operative orthoptics, surgery, and post operative orthoptics.

If any of these are improper, or incomplete, success in treatment will be more difficult. Success can only come through intelligent integration of all of these steps.

What is success? 1) 100% functional cure. 2) Some phoria but good fusion on synoptophore but only fair fusion otherwise (with red

glass or Worth 4 dot test). 3) Small tropia with only fair fusion on the synoptophore. 4) Cosmetic only.

Failure to obtain success may be due to: 1) Improper diagnosis. 2) Improper treatment. 3) Vertical imbalance. 4) Poor cooperation on part of patient or parents.

Equipment necessary: 1) Pin in pencil eraser. 2) Prism bar. 3) Square prisms. 4) Muscle light (distance, near). 5) Worth 4 dot test (distance, near). 6) Millimeter ruler. 7) Cover (old x-ray film cut to size). 8) Synoptophore or other amblyoscope. 9) Doll. 10) Glasses for doll. 11) Gun flashlight. 12) Red glasses. 13) Refraction equipment.

I. History:

Take from the parents and ignore the child.

Age of onset—Early onset is more apt to have amblyopia, suppression, and ARC.

Duration—Longer squint has persisted more apt to have amblyopia, suppression, ARC, and more difficult to treat.

Characteristics: 1) Convergent or divergent. 2) Periodic or constant. 3) Fixed or variable in amount (fixed and constant is more apt to have amblyopia, suppression, and ARC).

Precipitating cause:

Heredity—Family history is important since refractive errors are apt to run in families, and also, if there is a family history of squint the squint is more apt to have a mechanical factor.

Birth and development—Difficulty and length of labor, the use of forceps, the possibility of an eye injury important since they may have to explain a congenital amblyopia. Development important since progress not so favorable in a retarded child as in a normally developing one.

Previous Rx.—Glasses, exercises, etc. Previous exercises may make ARC worse, if improperly done. Previous surgery.

II. Examination:

1. a) Gain the confidence of the child. b) Period of play. c) Use doll and flashlight gun. d) Make a game of it. e) Play peek-a-boo with doll. f) Get down on floor with young children. g) No white coat. h) Several children at once makes them feel at home. i) Be cheerful even if the child isn't.

2) Vision with and without glasses: a) Snellen letter chart after 7 years or earlier if possible. b) Snellen illiterate E chart from 2½ to 7. c) Teach the use of the E. d) Fixation a good test under 3 years: Good and central, 20/70; Unsteady and central, 20/200-20/70; eccentric, 10/200.

Treatment of amblyopia: Occlusion, constant. Length of time at least 2 months even if no improvement. Be sure to rule out other

causes of poor vision. Results, usually good if occlusion is total and prolonged.

3) Refraction: a) Atropin cycloplegia: Costenbader, at any age. Here, up to 6 yrs. of age. b) Retinoscopy—Let child play with lenses; demonstrate on doll. c) Correction, nearly full if less than 3 D; 3-6 D. —Leave 1 D. uncorrected at first and fully Rx in 3–4 months; 6 D.—leave 1.50 to 2.00 D. uncorrected at first.

Amount of squint should be measured under cycloplegia with full correction at 20 feet. This will give you the mechanical factor since all accommodation is eliminated.

4) Amount of squint and motility: a) Rotations in 6 cardinal directions. b) Prism cover test with and without glasses, 20 feet, and 13 inches. 1) Mechanical factor. 2) Accommodative factor is the mechanical factor subtracted from the total squint as measured at 13 inches, without cycloplegia and without correction. 3) Measure the angle of deviation in the 6 cardinal directions. This will indicate if the squint is greater in one angle than in another and will often reveal an otherwise unsuspected vertical anomaly.—Without glasses at 13 inches. Vertical anomalies can be treated by prisms if small and surgery if large. Surgery should never be performed unless the vertical deviation is present in the primary position and never on a dissociated double hypertropia. c) NPC—measure in millimeters—use a white-headed pin with two dots on it or other small, interesting object. Will help determine whether interni or externi should be operated. Normal, not greater than 70 millimeters; should be closer to 50. d) Perimeter, Hirschberg, etc. There are four ways to use two eyes: 1) Fuse. 2) ARC. 3) Suppress. 4) Diplopia. e) Fusion: Good fusion is the difference between a cosmetic result and a functional result. Too often we are satisfied with 1) good vision; 2) good ocular alignment. Difficult to stimulate and encourage good fusion without orthoptic technician and synoptophore, but it is important to know what can be done.

a) Test for ARC: One test is use synoptophore and note difference between objective and subjective angles. Objective is obtained by alternately flashing pictures before each eye and moving arms until no movement is noted. Subjective is letting patient move the arms until the pictures are aligned. After image test of Bielschowsky. Red glass and light at 20 feet with prisms.

Treatment of ARC very important since failure to correct it will result in failure to perfectly straighten the eyes. Is strictly an office procedure.

Treatment is 1) Using synoptophore at objective angle and massaging, repeating at great length. 2) Occlusion. 3) Begin with 5 years old and when vision is 20/50 or better. 4) Before and after surgery.

b) Test for suppression: 1) diplopia difficult to elicit with red glass. 2) Check marks on slides—absent—on synoptophore.

Treatment: Occlude the non-suppressing eye constantly. Try to stimulate diplopia with red glass. Flashing a synoptophore. Bar reading at home.

c) Test for fusion: Two advantages to obtaining good fusion: first, eyes are better stabilized after surgery, and second, if fusion is present the individual will have a better chance of entering a field of livelihood in which depth perception is necessary.

First grade demonstrated by fish and bowl type of slide—superimposition.

Second grade demonstrated by the rabbit type of slide in which there is a large element common to both slides (rabbit) and smaller elements (tail or ears) common to only one slide.

Third is demonstrated by slides which give an idea of perspective.

Treatment: Before fusion can be treated ARC and suppression must be eliminated. Check frequently or they may come back. Always begin the training by stimulating fusion at the angle of squint before attempting to converge or diverge from that angle.

III. General Health:

Run down, fidgety, irritable, nervous child is not a good candidate for any type of treatment. Often summers at seashores or in the country will help these children more than anything. General health should be good.

IV. Parents:

Time spent explaining is worthwhile. Emphasize mechanical factor and accommodative factor. Talk about weak and strong muscles, good vision in each eye, and teaching two eyes to see together.

Emphasize a great deal of time is required, and good cooperation is necessary.

V. Summary:

1) Accommodative convergent squint: a) Amblyopia usually not great. b) Suppression and ARC usually not present but not difficult to treat. c) Usually moderate to high hyperopia and must wear full correction. d) Occlude the amblyopic and suppressing eye. e) Dissociate only after obtaining a good result with glasses. Explain how this is done. f) No surgery.

2) Mechanical convergent squint: a) Amblyopia usually marked except in alternaters, and suppression and ARC are deep-seated. b) Therefore prolonged constant occlusion, stimulation of the maculae at the squint angle and fusion training at the squint angle are necessary. c) if NRC can not be obtained in 8 weeks surgery should be performed anyway since after surgery ARC frequently changes to NRC. d) Surgery always necessary in these cases.

3) Combined squint: a) Analyze carefully. b) Treat the accommodative factor first and then the mechanical factor.

Management of Divergent Strabismus

a) Divergence excess: 1) Refract—do not correct hyperopia; correct myopia fully. 2) amblyopia and ARC no problem. 3) Surgery—bilateral recession of external recti almost always indicated. 4) Convergence training if indicated.

b) Convergence insufficiency: 1) Amblyopia, suppression, and ARC are no problem. 2) Improve general health. 3) Improve fusion and increase convergence amplitude. 4) Rarely surgery.

c) Combined—correct b. first, then a. *Author's abstract.*

Surgical Technics in the Management of Motor Anomalies. *William B. Clark, New Orleans, La.* Presented at the American Academy of Ophthalmology and Otolaryngology, 1949.

The surgical management of strabismus begins before operation, with the making of an exact diagnosis, accomplished by 1) measuring the defect with square prisms and the screen comitance test in the primary position for both distance and near and in the six cardinal positions of gaze, with the record kept in prism diopters; 2) measurement of the defect on the major amblyoscope when an athoptic technician is available; 3) checking the defect on the perimeter, particularly if the error is an unusual type and seems to vary; 4) determination of the near point of convergence; 5) determination of the effect of glasses on the defect, this determination including the use of prisms, either separately or in combination with glasses; 6) determination of the fixing eye in all quadrants and of the behavior of both eyes behind the screen on the screen comitance test.

Only after all of this information has been recorded and evaluated is the surgical procedure planned. The ophthalmic surgeon must work out for himself, on the basis of his experience and individual techniques, the amount of resection or recession of any individual muscle in correlation with the degrees or prism diopters of variation. Two principles are important, 1) never to cripple any single muscle excessively, and 2) not to attempt to secure more than 20–25 prism diopters' correction by operation on any single muscle, more than 40–50 prism diopters' correction by operation on any two muscles, or more than 70–75 prism diopters' correction by operation on any three or more muscles. Adherence to these principles will leave undisturbed conjugate lateral movements, which are essential for a good functional result. A full knowledge of Tenon's capsule and its fascial relationship to the muscles and check ligaments is also essential. Rotation of the eye into the six cardinal positions of gaze after the patient has been anesthetized is a simple way of determining over activity or limitation of motion arising from ligamentous abnormalities.

Complete tenotomy is no longer widely popular. The incomplete

procedure, in which the tendon alone is cut, is safer but is also not widely used; the results depend upon the strength or weakness of the lateral ligaments, which are preserved. Button-hole tenotomy is useful, particularly when shortening a paretic muscle, especially in the inferior rectus, if it is combined with weakening its direct antagonist by a small amount.

The most popular and useful operation on the medial and lateral recti is the Jamison recession operation, performed by the classical technique, but with closure in layers and with the substitution of catgut for silk sutures. The inferior oblique, as Fink's excellent cadaveric dissections make plain, is best attached in the region of its scleral insertion; when this procedure is combined with a lengthening or shortening operation on the lateral rectus, the technique is simple and the results are good. The normal relationship of the inferior oblique muscle to the globe should be maintained. Beeke's studies have simplified operations on the superior oblique. The lengthening (weakening) operation gives satisfactory results without great difficulty, but a shortening procedure is almost impossible to perform without taking off the superior rectus tendon to provide better exposure.—*Author's abstract.*

Cornea, Sclera and Tenon's Capsule

See Contents for Related Articles

Retina

Pathology of Hypertensive Retinopathy. *Michael J. Hogan, San Francisco, Calif.* Presented at the American Academy of Ophthalmology and Otolaryngology, 1949.

Richard Bright, in 1836, first described the visual defects which occur in nephritis. Following the invention of the ophthalmoscope, numerous observers described the retinal and optic nerve lesions which occur in hypertension and nephritis. The anatomic lesions which occur in the retina and optic nerve were aptly described by Turck, Virchow, and Liebreich. The ocular lesions originally were described under the term, albuminuric retinitis, and it is only in recent years that protests against the use of the term, "albuminuric retinitis," have begun to be accepted. The following objections may be made to the use of the title "Retinitis":

1. The retinal and optic nerve lesions are not inflammatory in nature.

2. It is possible to have the fully developed clinical picture of "albuminuric retinitis" without evidence of any involvement of the kidneys.

3. An advanced chronic glomerulonephritis may be present, with albuminuria and uremia, and there may be no evidence of ocular involvement.

Pathogenesis: The retinal changes which occur in hypertension and

nephritis depend essentially on the development of hypertensive vascular lesions plus an unknown toxic factor which is superimposed upon the vascular lesions.

Correlation of Ophthalmoscopic and Pathologic Changes in Hypertension and Nephritis: In general there are four types of changes to be observed: I. Vascular changes. II. Edema. III. Exudates. IV. Hemorrhages.

I. Vascular Changes:

A. Narrowing:

1. Generalized narrowing.

Ophthalmoscopic findings: The ophthalmoscope reveals generalized attenuation of all the retinal arterioles.

Pathologic changes: Frequently there are no histologic changes early in generalized narrowing. The narrowing is due to an increased vascular tonus. If the increased tone of the vessel persists, medial and intimal sclerosis occurs.

2. Localized narrowing.

Ophthalmoscopic findings: In the active stage the vessel at the site of spasm is less distinct. At the later stages, when the local narrowing becomes fixed, the outlines become more distinct.

Pathologic changes: Early there may be no histologic changes; later the picture of localized sclerosis of the vessel wall, with permanent narrowing, occurs. The narrowing may be due to changes in the wall itself or to narrowing of the blood stream by an endarteritic process.

B. Sclerosis.

1. Loss of the translucency of the vessel wall.

Ophthalmoscopic findings: The transparency is lost and veins cannot be seen through the arterial wall.

Pathologic changes: The loss of transparency is due to fibrosis of the intima and media of the vessel wall.

2. Alteration in the vascular reflex.

Ophthalmoscopic findings: The normal vascular reflex is a central bright streak caused by reflection of light from the blood column and from the media of the vessel wall. Three types of alterations are noted in the vascular reflex:

a) Copper-wire arteries: Ophthalmoscopic findings—The reflex is broader and softer and coppery in appearance. Pathologic changes—There is thickening and hyaline degeneration of the media of the arteriole.

b) Silver-wire arteries: Ophthalmoscopic findings—The entire vessel reflects light homogeneously and the vessel appears as a bright, white streak, in the retina. Pathologic changes—There is marked hyaline degeneration of the whole wall of the arteriole associated with large amounts of lipoid material.

c) Irregularity of the reflex: Ophthalmoscopic findings—Beading,

dots, or increased brightness in localized areas may be noted irregularly dispersed along the arterioles. Pathologic changes—These changes are due to local hyaline changes in the wall, resulting in the irregularity of the lumen. Localized spasm is thought to account for some of these changes.

3. Sheathing of the vessels, ophthalmoscopically, is seen near the disc as a normal finding. It is due to an accumulation of glial tissue and there are several types of sheathing encountered.

a) Parallel sheathing ophthalmoscopically occurs as a white line along the blood vessel wall. Pathologic finding—Sheathing is due to changes in the subendothelial connective tissue, where there is proliferation. The media may be hypertrophied and the adventitia thickened.

b) Pipe-stem sheathing: Ophthalmoscopically the whole wall appears to be invested and appears as a white fibrous cord. The sheathing first appears at the arteriovenous crossings or where a vein and artery are parallel. Pathologically pipe-stem sheathing is merely an advanced stage of parallel sheathing, in that the intimal, medial, and adventitial changes extend around the whole vessel wall and render it opaque. Sheathing alongside veins commonly is due to large phagocytic cells which are removing debris and lipoid from a damaged retina.

4. Arteriovenous crossing changes.

a) Constriction of the veins: Ophthalmoscopically there is usually no change in calibre of normal veins at the crossings. During the process of sclerosis the veins appear constricted, and appear partially covered, and eventually dilate beyond the crossing (Gunn's sign). Pathologically, the invisibility of the vein beneath the artery is due to sclerosis of the arteriolar wall which renders it opaque. A process of phlebosclerosis may also occur which extends out into the wall of the vein beyond the crossing, rendering the vein further less visible.

b) Deflection of the vein: Ophthalmoscopically the vein venous crossing becomes more near a right angle (Salus' sign). Pathologically the alteration in the course of a vein is due to the fact that the vein and artery possess a common sheath at the crossing and the arteriosclerotic process extends into the wall of the vein. With sclerosis of the artery it elongates and drags the vein with it, causing a change in the course of the vein.

5. Irregularity of the lumen: Ophthalmoscopically the arterial lumen may show widenings and narrowings and these may be generalized or localized. Pathologically in larger vessels these changes are due to a patchy proliferating endarteritis and, occasionally, are due to a localized sclerosis of the perivascular adventitial tissue or to localized spastic contraction.

6. Tortuosity of the Vessels: Ophthalmoscopic findings: A certain degree of tortuosity is normal. This sign must be regarded with suspicion. Extreme tortuosity of the small macular twigs is a valuable sign

of sclerosis, associated with hypertension. Histologically the hypertrophic thickening of the smaller arteriolar walls is seen which causes elongation of the arteriole, resulting in its tortuosity.

7. Generalized attenuation: Ophthalmoscopically this is usually a sign of hypertension. Pathologically in sclerosis narrowing of the vessel may prove to be due to intimal hyperplasia.

II. Edema:

Ophthalmoscopically one of the first signs of decompensation of the retinal circulation is the development of edema. In the beginning the edema fluid is not visible in the retina. As coagulation occurs, this fluid concentrates and is recognizable with the ophthalmoscope. It appears as sharply defined exudates with round or irregular shape; they vary in size from pin-point to one-half disc diameter in size. They lie deeper in the retina beneath the blood vessels and are frequently seen between the disc and macula or surrounding the macula, but they may be widely scattered throughout the posterior segment. Pathologically the changes in edema depend on exudation and degeneration. The edema fluid is diffusely spread through the retina but has a preference for the nerve fiber layer and internuclear layers. The capillaries dilate. Fluid diffuses through the walls. Later a fibrin-rich plasma is deposited and red blood cells may pass through the capillary walls. Some zones in the retina show destruction of all elements. Others show fibrin and lipoid deposits in the neural and glial tissues. The intranuclear and other layers are loaded with phagocytes.

a) The star figure: Ophthalmoscopically the star-figure results from concentration of the edema exudates around the macula. Pathologically the star-figure is formed by the deposition of hyaline and lipoid deposits which are arranged in groups along the radiating fibers of Henle in the retina.

b) Papilledema: Ophthalmoscopically edema of the disc may be faint to marked in amount and extend into the peripapillary region. The disc is more apt to be pale in nephritis due to the secondary anemia. Pathologically the nerve-fiber layer exudates are frequently due to varicose swellings of the nerve fibers which form cytoid bodies. The principal edema of the disc and adjacent retina is due to accumulation of fluid between the nerve fibers.

III. Exudates:

Ophthalmoscopically exudates occur in most cases which have an abrupt onset, a stormy course, and exhibit toxemia. Cotton-wool patches are seen in this type of case. The cotton-wool patches are soft and occur around, under, or over the blood vessels. They may completely surround the disc. They have a strong tendency to resolve rather rapidly. Histologically cotton-wool patches are due to a diffuse deposition of fibrin to form a dense network throughout the portions of the retina where there are loose tissues, especially the inner layers.

IV. Hemorrhages:

Hemorrhages are round or linear in shape, vary in size, but are generally small. They appear at all levels in the retina and are most marked in areas where the vessels are affected. They change constantly in number and size. Histologically hemorrhages are capillary in origin and have a preference for the nerve-fiber and internuclear layers. Deeper hemorrhages are round, and linear hemorrhages are found in the nerve fiber layer.

Clinical and Histologic Findings in Malignant Hypertensive Retinopathy

Ophthalmoscopic findings: Malignant hypertension is characterized by extreme papillary and peripapillary edema associated with narrow attenuated arteries, diffuse superficial hemorrhages, cotton-wool exudates. The exudates may be large and numerous, forming large peripapillary ring or macular star. Later the exudates become hardened and more sharply delineated. Histologically the hemorrhages, exudates, and vascular changes are similar to those described above. An additional finding is the occurrence of arteriolonecrosis of the walls of vessels in the retina and choroid.

Changes in the retinal vessel walls which are easily recognizable ophthalmoscopically, occur in subdivisions far beyond the first divisions. There are no media and adventitia in these subdivisions so that it is not correct to discuss pathological changes as one would in arterioles elsewhere.—Editor.

Heredodegenerative Diseases of the Retina. *Herman Elwyn, New York, N. Y.* Presented at the American Academy of Ophthalmology and Otolaryngology, 1948.

There are two terms commonly used to indicate certain progressive diseases; (1) abiotrophy, and (2) heredodegeneration. The term "abiotrophy" was first used by Gowers. He found the word "biotrophos" which had been used by an ancient writer in the sense of "vital nutrition." By prefixing the negative particle he coined the word "abiotrophy," meaning the absence of vital nutrition. Gowers used this term for diseases characterized by "a degeneration or decay in consequence of a defect of vital endurance."

The term "heredodegeneration" was used by the Hungarian neurologist Jendrassik in the same sense as the term "abiotrophy" was used by Gowers. The term "heredodegeneration" is preferable, and is generally used for those hereditary diseases, especially of the nervous system, which are characterized by the progressive loss of a certain tissue. It is used also for those hereditary diseases which are characterized by a progressive loss of elements of a certain tissue, such as ganglion cells or glia cells.

There is a third term used by the neurologist Kehrer, namely, "heredoconstitutional." He uses the term for certain diseases in which a hereditary vital defect exists but there is no progressive loss of tissue and the disease remains stationary for many years and perhaps throughout life. I shall use these two terms: heredodegenerative and heredoconstitutional.

Among the diseases of the retina are a number which must be classified as heredodegenerations. They have these facts in common: They are bilateral; they are hereditary and familial; they are not due to any external cause, to inflamation or to vascular changes; with the exception of the heredoconstitutional diseases, they are progressive. Because the elements of the retina are neural or of neural origin, they frequently partake of the heredodegenerative diseases which affect similar elements in the central nervous system. An attempt to classify the heredodegenerative diseases which affect the retina must take into account the fact that the retina is made up of a number of separate elements and that each of these elements is theoretically subject to some form of heredodegeneration. A review of the various diseases of the retina shows that this is actually the case and that the degenerations of the retina can be classified according to the individual layer or kind of cells primarily affected. The layers of the retina thus subject to some form of heredodegeneration are: (1) The elastic layer of Bruch's membrane. This membrane, although not a part of the retina, is a border membrane forming a barrier between the choroid and the retina. It is subject to degeneration which has consequences for the retina. (2) The pigment epithelium. (3) The neuro-epithelium. (4) The nuclear layers. (5) The ganglion cells. (6) The nerve fiber and the glia cells.

The individual heredodegenerative diseases of the retina involve primarily certain retinal elements and can be classified as follows:

A. Originating in the elastic layer of Bruch's membrane.

1. Disciform degeneration of the macula (Kuhnt-Junius type)

2. Angioid streaks in the fundus, occurring: a) alone; b) in association with pseudoxanthoma elasticum (Groenblad-Strandberg syndrome).

B. Originating in the pigment epithelium.

1. Hyaline or colloid bodies (drusen) of the basal layer of Bruch's membrane.

C_1. Originating in the neuro-epithelium (1) In the central, or macular, area.

1. Heredodegeneration of the macula, including the infantile, juvenile, adult, presenile and senile types, occurring: a.) alone; b.) in association with: i) degeneration of the peripheral neuro-epithelium; ii) atrophy of the optic nerve; iii) color blindness; iv) mental deterioration.

C_2. In the extramacular neuro-epithelium.

1. Retinitis pigmentosa, occurring: a) alone; b) in association with:

i) degeneration of the macula; ii) color blindness; iii) deafness; iv) mental deterioration; v) the juvenile type of amaurotic family idiocy; vi) the Laurence-Moon-Biedl syndrome.

C_3. Heredoconstitutional diseases of the neuro-epithelium

1) Retinitis punctata albescens

2) Congenital night blindness: a) without changes in the fundus; b) with grayish white discoloration of the fundus (Oguchi's disease)

3) Color blindness.

C_4. Heredodegeneration of doubtful origin but standing in some relation to degeneration of the neuro-epithelium; 1) Gyrate atrophy of the choroid and retina; 2) Choroideremia, a heredoconstitutional disease;

D. Originating in the ganglion cells; 1) Infantile type of amaurotic familial idiocy (Tay-Sachs disease) affecting only the ganglion cells; 2) Juvenile type of amaurotic familial idiocy involving the neuro-epithelium in addition to the ganglion cells; 3) Niemann-Pick disease.

E. Involving the nuclear layers; 1) Peripheral cystoid degeneration in senile and presenile eyes; 2) Widespread cystoid degeneration, forming the basis for rupture of the retina with hole formation and consequent retinal detachment; 3) Circinate retinitis.

F. Involving the nerve fiber layer; 1) Tuberous sclerosis, a widespread heredodegenerative disease causing the production of abnormal cells and tumors in this layer.—*Author's abstract.*

Macular Lesions. *Ralph I. Lloyd, Brooklyn, N. Y.* Presented at the American Academy of Ophthalmology and Otolaryngology, 1949.

Trauma

Usual history is blunt trauma causing sudden compresion of globe. In penetrating wounds the damage is so severe that the macular condition can be determined only by finger counting, light projection, etc.

Rupture of choroid is associated with damage to retina over a larger area than the tear would indicate. Red-free light gives an excellent idea of the condition of the macula.

Amotio Retinae. The macula may show decided change after trauma but return to normal is possible if there are no hemorrhages. This is of medicolegal importance.

Holes in macula occur after trauma and spontaneously in myopes and older patients. The blow may not be severe. The visible lesion is in the choroid and the vision may be very good.

Massive Retinosis is seen after injuries with considerable hemorrhage. The organization extends into the vitreous with serious loss of vision but the eye may retain integrity for years. Ablatio falciformia is always connected with the disk and lacks the fine terminal brush work

in the vitreous. Exudate may organize about foreign bodies and is easily mistaken for ablatio as well as retinosis.

Late macular changes after trauma may be visible only after some weeks but the loss of vision is immediate. Careful testing is necessary to decide.

Purtscher's Edema is the effect of venous stasis, as in compression of chest by steering wheel of an overturned auto. The fundus is characterized by white clouds and hemorrhages. The prognosis is better in cases with less hemorrhage. A central scotoma may result with very little visible evidence.

Photo trauma includes eclipse scotomas, effects of electric flashing, lightning, exposure to infra-red, ultraviolet, and x-rays.

Warnings to avoid direct observation of the sun date far back in human history, but each eclipse adds new cases to the literature. After the eclipse of 1912, Cords reported nearly 400 cases in Germany. The macular changes are rarely pronounced but observers agree that objective evidence is always seen. Prognosis is good but permanent damage may result.

Electric flashing rarely produces permanent damage because preventive measures are in common use. Less severe damage from strong light and mirrors was common in the days of arc lighting. Corneal ulcers, injection, and lacrimation with intense photophobia were usual but prompt recovery followed ordinary care. Annoying asthenopia follows the use of glass-topped desks because of reflection. Modern fluorescent lighting may be quite troublesome if not carefully arranged. The cataract of glass blowers is a rarity today but during the war furnaces were set up without the necessary protection to the workers about them. Snow blindness is noted in Xenophon's "Anabasis" but even the Eskimos know how to fashion protective devices. After cataract operations red or blue vision occurs and is a mild form of overexposure but rarely causes trouble.

Diseases with Macular Lesions as Prominent Features

Fuch's Spot. A pigmented spot in the macula of high myopia with hemorrhages, occurs also in low degrees and without hemorrhage. In the original group lowest age was 16, the average 42. Both eyes are involved in many instances and no satisfactory explanation has been offered. Lehmus made a microscopic examination of a previously studied case, finding the lamina of Bruch and choroid little affected. Pigment epithelium had proliferated and was heaped in masses equal to thickness of choriocapillaris. Pigment disappeared from the periphery with a mass of acellular fibrin superimposed adherent to retina. Lesion produces a central scotoma. Fuchs: Ztschr. f. Augenh., 5:171, 1901.

The Senile Macular Changes. Atrophy of the choroid with sclerosis of the choroidal vessels is common in the eyes of older persons, but has

very moderate effects upon vision other than contracted fields and enlarged blind spots. The process begins usually in the periphery and macular involvement is late. From the appearance of the exposed deeper choroidal vessels one would expect more serious effects. This is explained by Verhoeff and Marin Cohen, who found marked changes in the vessel walls but unaltered calibers.

When the macula is involved there is a sheen or watered silk effect over the macular area, the background is "meaty" and the finer vessels undulate. Changes in the macular retina can be seen with red-free light when the vision begins to fail. While most patients escape serious effects, a series of lesions may follow with the same basic pathology. These changes include hole in the macula, pseudotumors which later may become Coats's disease or disciform degeneration of the macula, and patches of choroidal atrophy exposing what looks like sclera but is organized tissue. Angioid stripes are regarded as a definite entity with very similar pathology.

The basic pathology of this senile group is traced to the lamina of Bruch, allowing a serous or hemorrhagic exudate to strip the pigment layer of the retina from the lamina of Bruch, to organize later to form the cotton-wool masses of Coats's disease or the firm tissue seen in disciform degeneration. Choroidal hemorrhages and retinitis circinata are parts of the picture.

Progressive Choroidal Atrophy is a term of convenience for cases of advanced atrophy with a patch of approximately normal choroid in the macular area. We have the unique picture of an approximately normal macular area surrounded by exposed, heavily pigmented deep choroid with sclerosed vessels. When the macular patch of choroid disappears, the patient is blind. The typical case does not complain much of night blindness but there is a group occurring in older patients with good vision in good light, ring scotomas but optic disk and retinal vessels unaltered. This type has been referred to as progressive choroidal atrophy but the term choroideremia is also used (see Choroideremia).

Pseudotumors of the macula have been often diagnosed as choroiditis because the modest elevation is easily missed with the monocular instrument. First complaint is blurred vision which may precede objective evidence. Small hemorrhages in the macula, which becomes lighter in color and somewhat raised, are common early signs. The mass may recede with improvement in vision but more often it becomes more prominent. Some have remained in this stage for months but as a rule changes follow in a few months.

If the clear exudate breaks through the pigment layer of the retina and is seen through the retina only, a type of cyst is seen which may be explored with the binocular scope. Either the original lump or the cyst may undergo further changes. The exudate may organize to form the

cotton-wool patches of Coats's disease, it may organize into a firm subretinal layer of disciform degeneration of the macula (Kuhnt-Junius), or it may spread laterally raising the retina in folds making a type of flat detachment over a limited area. The organized exudates may form bone, cartilage, connective tissue, and cysts. Throughout the process, hemorrhages and circinate retinitis are common. Pseudotumors are not always the early stage of Coats's disease, disciform degeneration, and advanced atrophy of the tapetum in the macular area, but they are easily overlooked unless the binocular scope is used. The initial lesion may be hemorrhagic, in which case details of the mass are lost and the similarity to "choroidal detachment" after cataract operation is striking.

The primary lesion of pseudotumor is an accumulation of fluid beneath the pigment layer setting off a series of characteristic changes in the pigment epithelium, with the retina changed but little. Behr assigns to the lamina of Bruch control of nourishment to and waste products from the outer retinal layers. Elastic tissues of the body pay toll to time well in advance of other structures and he believes the first change affects this membrane, allowing an accumulation of fluid beneath the pigment layer which reacts much as the lens stroma reacts to exposure to aqueous. Proliferation and degeneration follow, forming a sort of capsule, thicker at the borders, giving the ophthalmoscopic impression of circinate retinitis (Seefelder). Behr asserts that formation of connective tissue, cartilage and bone is a genuine metaplasia, but a more reasonable explanation has been offered by Verhoeff, Axenfeld, Sandoz, Vogt, and others. They demonstrate breaks in the lamina through which capillaries pass from the choriocapillaris to the subretinal mass. This invasion by vessels would also explain the frequent hemorrhages and new vessel formations seen in these cases.

The lesions are often bilateral with an interval between attacks. The patients are of the older group but typical cases have occurred in people in their thirties and even younger. The patients are usually of the "healthy arteriosclerotic" group, but syphilis occasionally appears in the case history. In young patients tuberculous lesions are very similar in appearance at first but the vitreous is involved very early and there are signs of healed lesions from previous attacks. Post traumatic hemorrhages into the vitreous may organize and leave permanent masses projecting from the disk which should be easily differentiated. There are no congenital defects of the macula which should confuse. Metastasis from breast or prostate gland will produce a mass indistinguishable from pseudotumor but which rarely affects the macula.

Central Retinal Retinopathy. Because of rarity we are in doubt as to important details. The few coming under observation have shown a retinal edema with dots and fine spots over an elevated tapetum. With the hand scope they appear as a lenticular mass anterior to the

retina but the binocular scope gives quite a different picture. None has been examined in the laboratory.

Angioid Stripes have been regarded as an entity, but thanks to Bock, Hagedorn, Klien and Winkelman, we know that the pathology of this condition is similar to that of pseudotumor. It is often seen in families along with pseudoxanthoma elasticum, Paget's disease, sclerotic patches in the elastic lamina of the ciliary vessels outside of the globe, and in other arteries of the body. The lamina of Bruch splits. There are lime deposits in the outer layer and breaks with and without changes in the pigment layer over them. Hagedorn and Bock have plotted the stripes over a mapping of the breaks in the lamina showing the intimate relation between the two. The late stages resemble those of pseudotumor with what looks to be a patch of sclera but is really a mass of organized tissue (disciform degeneration of the macula).

Hereditary Macular Degeneration

Our knowledge of these diseases is incomplete because certain types do not furnish laboratory material and very few affected families have been observed over an extended period. The characteristics are:

Hereditary and familial incidence.

Bilaterality.

Onset—birth, second dentition, puberty, end of skeletal growth, beginning of involution, senility.

Time and manner of onset are the same for the members of a given family.

No single characteristic fundus picture but type for a family runs true.

If transmitter is a female she is usually affected, but male transmitter escapes.

There are four general types but classification is subject to change as we learn more about the various types and phases of development.

Type 1. Colloid degeneration of Bruch's membrane is the most common form and least harmful. Vision is not affected even in most pronounced cases until later in life when pigmentation and atrophy of the tapetum begin in the macular region. The youngest case seen was a 5 year old boy but it usually appears after the fourth decade in patients who come regularly for refraction. Most are discovered during routine eye examinations. In the literature this group appears with the title of choroiditis guttata, honey-combed choroiditis, etc. The lesions may be few or innumerable, limited to the macular region or widespread, as light-colored or white spots of various sizes and shapes but usually round. Treacher Collins made the only microscopic examination. The lamina of Bruch is thickened with nodules of hyaline material, with little effect upon the outer layers of the retina except by pressure. Drusen bodies concern the same membrane but are different in form. The

lesions are seen with the binocular scope in the tapetum, and the integrity of the macula shown in red-free light.

Type 2. Retinitis punctata albescens is featured by fine white dust or discrete white dots. The first type is most pernicious and the vision is affected early. Ring scotoma, night blindness, and color blindness are some of the symptoms, and blindness the final state. Lauber suggests the name "fundus albipunctatus cum hemeralopia congenita" for a milder type with discrete white dots in the periphery, mild night blindness, moderate contraction of the fields, and some loss of vision. Both types are rare and neither has furnished laboratory material.

Type 3. Maculocerebral degeneration (infantile and juvenile) has been well studied as both types lead to hospitalization. The infantile form described by Tay and Sachs is well known for its cherry red spot in the fundus and rapid decline and death of an infant born in good condition.

The degeneration involves the ganglion cells of the retina, optic thalamus, and cerebellum. Juvenile forms begin with the fine pigment in the macula and atrophy of the tapetum behind the macula. The vision may suffer well in advance of objective evidence and is much poorer than the changes would seem to justify. Inheritance may be sex-linked or not. If the onset is before puberty, the patient usually passes into idiocy and dies. If the onset is after puberty, the cerebral changes may not occur. The course of the fundus changes is much like that of retinitis pigmentosa, with the difference that the latter does not have macular changes.

Microscopic examination of the juvenile forms has been made in institutions for care of such cases. Paton and Holmes report "loss of vision, atrophy of disc, tapetoretinal degeneration, shrivelled vessels" as the ophthalmoscopic changes and "degeneration of outer retinal layers, with replacement by proliferated neuroglia." Inner retinal layers were but little affected. Also "atrophy of cerebellar cortex, disappearance of granular layer, diminution of Purkinje cells and their fiber network."

Type 4 is featured by atrophic lesions as if some layers of the choroid were missing with pigment reaction. The vision is not affected in the early stages. This lesion may be mistaken for some types of extrapapillary coloboma. None of these has been examined in the laboratory.

Choroidermia

This term was introduced by Mauthner, who believed the case he reported was a congenital deficiency of the choroid. In the literature only 3 cases may rightfully be considered as such (Conner, Cogwill, Landman) with an arrangement of blood vessels that could not be caused by disease. Other cases reported as atrophy of the choroid or choroideremia (Parker and Fralick, Wolf, McCulloch and McCulloch, Ester-

man, Carroll) are definitely progressive abiotrophies occurring in males while the female transmitter (if found) retains useful vision with a fundus picture much like that of inherited chorioretinitis.

A third group has been studied by Verhoeff who explains the macula against a white background in cases of pigmentary retinitis as a glial overgrowth obscuring the choroid and its vessels. Bahn observed a case which, when first seen, was typical of this form, but in ten years the obscuring tissue had disappeared exposing the choroid and its sclerosed vessels. This is an infrequent type and binocular ophthalmoscopy will show much pigment and choroidal vessels not seen with the hand scope. Fourth group appears in the literature as progressive choroidal atrophy (Morton). The patients are older persons with a patch of fairly normal choroid in the macular area surrounded by heavily pigmented deep choroid with sclerotic vessels. Vision is surprisingly good in good light with ring scotomas. Disk and retinal vessels are changed but little. One is amazed at the moderate loss of vision with such severe changes. These are extremely rare and there are few laboratory reports. The term, choroideremia, has confused the literature because it is generally assumed that it must be congenital. Until the pathology and details of these cases are better known, the terms congenital and acquired choroideremia should be used.

Vascular Lesions

Vessel communications are incompatible with good vision unless congenital. Postnatal communications may be a fortunate outcome in retinal hemorrhages. Supplying return connections are usually venous and composed of very fine network. The coarse communications allow arterial blood to flow into the veins without reduction of pressure, resulting in loss of characteristics of both artery and vein. This is seen in angiomatosis retinae and after blunt trauma. The eye is lost as a result of glaucoma.

Occlusion of Central Retinal Artery. This is precipitated by heavy lifting or endocardial disease.

The central vein and artery are both involved in changes of age and arteriosclerosis. The occluding lesion may canalize, but retinal tissues cannot recover from obstruction of circulation unless a ciliary vessel escapes, in which case vision may improve decidedly.

Diagnosis of central occlusion after a period of time is not an easy matter, being mistaken for optic nerve atrophy, which is a part of the picture. Menace of central vein blocking is glaucoma.

Macular Hemorrhages are a part of angioid stripes, senile macular disease, diabetes, tuberculosis, angiosclerosis and blood diseases. The large hemorrhages are usually choroidal in origin. Not a few of these cases are without explanation.

The definite stripe of angioid disease is sometimes missing in a family with typical cases. Hemorrhages may be anticipated if minute enlargements are found on some of the minute macular vessels. Effort has been made to link these with Buerger's disease but without success.

Subhyaloid hemorrhages are located beneath the internal limiting membrane (Vogt).

Preretinal edema is extremely rare. Only two accumulations of fluid anterior to the retina have been under observation. An edema of the retina as a part of pseudotumor of the retina is much more common.

The binocular scope will demonstrate the elevation of the tapetum. 52 references.—*Author's abstract.*

Neuro-Ophthalmology, Optic Nerve, Visual Pathways, Centers and Visual Fields

Differential Diagnosis of Primary Optic Atrophy. *H. Saul Sugar, Detroit.* Presented at the American Academy of Ophthalmology and Otolaryngology, 1949.

We have all been taught to distinguish between two types of atrophy of the optic nerve, one, a primary affection and the other, secondary to an antecedent inflammation. This division into primary and secondary types is simply descriptive of the appearance of the nerve head and does not have real etiologic significance since the appearance of the nerve head depends on whether the lesion is located at the nerve head or somewhere higher up the third neurone pathway. An inflammatory lesion at the disc will produce a secondary atrophy while the same lesion a little way up the nerve will give an apparent primary atrophy.

From a purely descriptive point of view it is possible to distinguish five types of nerve head changes as they occur in optic atrophy:

1) Ascending atrophy—secondary to: a) Chorioretinal disease; b) Arterial occlusion; c) Amaurotic idiocy.

2) Postneuritis atrophy—Here the nerve is greyish white with blurred margins. The cup is filled. There is old exudate along the vessels. A peripapillary pigment disturbance may be present. The arteries are usually thinned.

3) Primary or simple atrophy. The disc here is white with a sharp, normal margin. The lamina cribrosa is visible. There is shallow cupping often. The vessels are unaffected usually.

4) Glaucomatous atrophy. The characteristic picture here is excavation of the disc with bending of the vessels at the margins.

5) Temporal atrophy.

Optic atrophy may better be classified from an etiologic viewpoint. (Modified from Duke Elder.)

I. Ascending consecutive atrophy—due to destruction of the retinal ganglion cells.

A) secondary to retinal or chorioretinal disease.

1. postinflammatory—after diffuse chorioretinitis.

2. degenerative: a) primary pigmentary degeneration—pigmentary retinitis; b) amaurotic family idiocy and cerebromacular degeneration; c) myopia.

B) secondary to circulatory disturbances of retina.

1. Occlusion of central retinal artery.

2. Post-hemorrhagic.

II. Sclerotic and nutritional atrophy.

1) Arteriosclerotic atrophy involving nutrient vessels of the optic nerve; 2) Anemias; 3) Avitaminoses (alcohol).

III. Pressure Atrophy.

A) Glaucomatous atrophy.

B) Post-papilledema atrophy.

C) Arterial pressure atrophy produced by sclerosed internal carotid, ophthalmic artery in the foramen, by a normal artery displaced by a pituitary tumor, or by aneurysms of the internal carotid.

D) Bony pressure in the optic foramen.

1) Osteitis deformans; 2) oxycephaly; 3) leontiasis ossea; 4) hydrocephalus; 5) craniofacial dysostosis.

E) Tumor pressure atrophy.

1) nerve sheath; 2) pituitary tumors; 3) frontal and temporosphenoidal tumors; 4) basal meningiomata; 5) neurofibromata.

IV. Post-inflammatory atrophy.

A) Post-neuritic atrophy, or post perineuritic atrophy.

B)Primary or simple (descending) atrophy—central nervous system disease.

1) Disseminated sclerosis; 2) Neuromyelitis optica; 3) Disseminated encephalomyelitis; 4) Herpes Zoster; 5) Lethargic encephalitis; 6) Tabes; 7) Dementia paralytica; 8) Hereditary ataxias.

V. Toxic atrophy—diabetes, methyl alcohol, arsenic, lead, atoxyl.

VI. Traumatic atrophy—avulsion of the nerve, skull fractures.

VII. Atrophies of unknown cause—Leber's.

The various clinical procedures which may be necessary in the differential diagnosis as indicated are:

1) Ophthalmoscopy; 2) Tonometry; 3) Exophthalmometry; 4) Visual fields; 5) X-rays of skull; 6) X-rays of optic foramina; 7) Encephalography and ventriculography; 8) Angiography of cerebral vessels; 9) Spinal puncture and studies of spinal fluid; 10) Serology; 11) Glaucoma provocative tests.

I. Ascending Consecutive Atrophy (due to destruction of the retinal ganglion cells).

A. Secondary to retinal and chorioretinal disease.

1. Post inflammatory.

The post-chorioretinitic atrophy is recognized ophthalmoscopically only when the damage to the ganglion cells involves a sufficient area. The discs here are sharply demarcated and the vessels remain normal.

2. Degenerative.

In the degenerative consecutive atrophy associated with pigmentary degeneration the disc is waxy and sharply outlined with marked vascular narrowing. The same picture may be present in syphilitic pigmentary degeneration.

In myopic atrophy, in addition to pallor of the disc, myopic crescent, myopic conus and circum papillary choroidal atrophy may be present. Cavernous atrophy simulating glaucoma may occur in myopia.

In the familial idiocy cases the ganglion cells are affected, resulting in ascending atrophy.

B. Secondary to circulatory disturbance of the retina.

1) occlusion of the central retinal artery; 2) post-hemorrhagic.

II. Sclerotic and Nutritional atrophy.

Arteriosclerotic atrophy, anemias, and avitaminoses. In sclerotic atrophy ischemia of the nerve presumably results from narrowing of the nutrient vessels supplying the optic nerve from the pia.

III. Pressure Atrophy.

A) Glaucoma is the most important cause of pressure atrophy of the optic nerve. Excavation of the nerve head commences at the temporal side of the disc and gradually extends. The vessels are shoved to the nasal side and characteristically hook around the edge of the nerve. There may be a glaucomatous peripapillary choroidal atrophic halo.

It may sometimes be difficult to distinguish from simple atrophy and from deep physiologic cupping as well as from coloboma of the nerve.

Pseudoglaucoma

This condition is a nonglaucomatous atrophy of the nerve with all the ophthalmoscopic characteristics of glaucomatous atrophy, and the visual field defects found in true glaucoma. I have seen such a picture in arteriosclerotic atrophy, descending atrophy from arterial pressure such as from pressure of sclerotic internal carotids, and after methyl alcohol poisoning.

It must be emphasized that pallor of the optic disc does not signify optic atrophy unless there is a demonstrable defect in the vision or visual field.

B. Post papilledema atrophy.

C. Arterial pressure atrophy produced by sclerosed internal carotid, ophthalmic artery in the foramen, by a normal artery displaced by pituitary tumor, or by aneurysms of the internal carotid.

D. Tumor pressure atrophy—nerve sheath tumors, pituitary tumors, frontal and temporosphenoidal tumors, basal meningiomata, neurofibromata.

E. Bony pressure in optic foramen—osteitis deformans, oxycephaly leontiasis ossea, hydrocephalus, craniofacial dysostosis.

F. Basal arachnoiditis.

IV. Post inflammatory atrophy.

Post-neuritic atrophy of the nerve is manifested by blurring of the disc margins, with a grayish white color of the disc. The cup is filled. There is old exudate along the vessels. The arteries are usually thinned.

"Primary optic atrophy" has been traditionally used to designate the atrophy occurring in tabes and dementia paralytica. It is generally conceded to be secondary to inflammation in the nerve and not to primary degeneration of the retinal ganglion cells. It is still termed primary, however, since the atrophy is not preceded by ophthalmoscopically-visible changes.

This type of parasyphilitic primary optic atrophy occurs in 6.5% of cases of tabes (Gowers) and 50% of juvenile tabes (Wilbrand and Saenger), 8–10% of parasyphilitic manifestations. In all of these, it usually appears 10–15 years after the primary infection, between the ages of 30–50, and in congenital cases the juvenile form becomes evident at about 10. Failure of vision and atrophy may be the first sign of tabes. It may precede other signs by 20 years or more. Other signs may be Argyll-Robertson pupils, absence of knee jerks, anesthesia (particularly the fifth nerve) and incoordination. It may, on the other hand, come on after the ataxia is well developed. The atrophy is unilateral at first, but the second eye is always involved. The discs are gray or white, with stippling of the lamina cribrosa, sharp margins and a normal surrounding retina. There may be slight atrophic cupping. The vessels are unaffected.

The symptoms in primary atrophy are loss of dark adaption, decrease in the color fields, photopsia and chromatopsia and field defects which are of three types: 1) Concentric contraction with late central loss; 2) Central or cecocentral scotoma; 3) Quadrantic or hemianopic defects.

Disseminated sclerosis follows an irregular course and rarely leads to complete blindness. It is often unilateral. The visual fields vary, with a central scotoma being relatively frequent.

V. Toxic atrophy: diabetes, methyl, alcohol, arsenic, lead, atoxyl.

VI. Traumatic atrophy: avulsion of optic nerve, skull fracture.

VII. Unknown causes: Leber's disease.

The Kestenbaum capillary number test has been found useful in evaluating optic atrophy ophthalmoscopically. With this test, the vessels which pass over the margin of the disc are counted. One starts at

the 12 o'clock point and counts separately the arteries, veins and small vessels which cannot be recognized as arteries or veins which cross the disc margin. Without pupillary dilatation, 9 large vessels (4 or 5 veins and 4 or 5 arteries) and about 10 small vessels are normally seen. In primary optic atrophy the number of arteries and veins remains the same but the number of small vessels is reduced to 7, 6 or even less, down to only 3.—*Author's abstract.*

Glaucoma and Hypotony

IV. Wide Angle or Compensated Glaucoma. *Samuel J. Meyer, Chicago, Illinois.*

In compensated glaucoma, the intraocular tension is, as a rule, higher than in the normal eye. While the circulation regulating mechanism of the intraocular fluids is normal in the latter, in compensated glaucoma there is a disturbance of the tension regulation factors. The glaucomatous eye reacts more readily to the influence of circulatory disturbances than the normal eye. A definite equilibrium of the intraocular fluid circulation is maintained so that acute rises in tension do not occur. Due to the absence of venous congestion, glaucoma simplex was designated as compensated glaucoma (Elschnig), compensated circulatory disturbance glaucoma (Wessely), or glaucoma lymphostaticum (Heerfordt).

The subjective complaints in the beginning of the disease are of a very mild character or may be entirely absent. Pain, cloudy vision and halos do not occur. The constant increased tension usually results in excavation and atrophy of the optic disk. Decrease in visual acuity causes the patient to present himself to the ophthalmologist for examination. If the visual decrease is very gradual, the patient may believe it due to the usual senile changes in old age and may further delay examination.

Externally, the compensated glaucomatous eye differs very little from normal because the changes in the anterior segment are negligible and can only be recognized with careful methods of technic. It is of the utmost importance to be fully conversant with the ophthalmoscopic picture of the optic disk and the functional changes which accompany the intraocular tension changes.

The diagnosis of wide angle glaucoma demands that a careful history be taken. It must include such factors as (1) the visual changes noted, if any; (2) headaches following the use of the eyes or otherwise; (3) fullness or tension in or about the eyes; (4) the presence of glaucoma in the family history.

Tonometry still constitutes our chief objective approach in diagnosis. One should become proficient in making accurate tonometric measurements, as there are many eyes in which only a slight rise in the

intraocular tension may be found long before any other objective findings or functional changes occur. The tonometry should be done at regular intervals during the waking period, as in the afternoon readings may be normal or subnormal and yet elevated in the early morning shortly after arising.

Optic disk changes occur later in the progress of the disease and may be suspected and looked for in individuals with large physiologic excavations which later become pathologically excavated to the disk margins, the temporal side being affected first. One should become familiar with the ophthalmoscopic picture of the normal disk, the physiologically excavated disk, and the pathologically excavated disk.

Visual field changes do not occur in the earliest stages, yet careful examination of the central fields upon a tangent screen may reveal an early enlargement of the blind spot both superiorly and inferiorly, the so-called Seidel's scotoma. This is later followed by an enlargement around the central area resulting in the arcuate Bjerrum's scotoma. Later on, Ronne's nasal step may develop either above or below, or both with a resultant loss of the entire nasal field.

Provocative tests should be employed early. The drinking test is probably the most efficient of the various tests for compensated glaucoma and consists of taking the tension before and after the patient has drunk two or three glasses of water. Any rise of 3 to 10 mm. Hg in the tension readings after drinking water should be viewed with suspicion.

Gonioscopy is of very little value in wide angle glaucoma as there are practically no changes in the early, and even later stages.—*Author's abstract.*

Gonioscopic Evaluation of Operations for Glaucoma. *Peter C. Kronfeld, Chicago, Ill.* Presented at the American Academy of Ophthalmology and Otolaryngology, 1949.

Evaluation of glaucoma operations is one of the many heavy responsibilities of the ophthalmologist. With all the methods which are now at his disposal, it is often impossible to determine to what extent a given operation affords protection again pathologic elevations of ocular tension. The data or methods upon which the ophthalmologist bases his decisions are: 1) Tonometric readings under conditions of ordinary life. 2) Careful visual inventories (chiefly central field studies). 3) Tonometric readings during provocative tests. 4) Inspection by microscopy and gonioscopy of the tissue alterations produced by the operation.

The method mentioned last has the obvious advantage of being fast and not too inconvenient. It is the purpose of this lecture to state the relationship between gonioscopic and external biomicroscopic appear-

ance of the area of operation, on the one hand, and the efficiency of the operation, on the other hand.

The Trephine Operation

Elliot's corneoscleral trephining is considered here as one form of corneosclerectomy. The most essential features of the gonioscopic appearance of a trephine operation are the location and the contents of the trephine hole. My associates and I have learned to recognize three typical positions, namely:

1. An anterior, mostly intracorneal position of the trephine hole characterized by a location of its posterior edge considerably in front of Schwalbe's line.

2. An intermediate position characterized by a location of the center of the trephine hole slightly in front of Schwalbe's line.

3. A posterior position characterized by a location of the center of the trephine hole with the trabecular area.

The three positions overlap to a large extent. The marked variations in the length of the anterior projection of sclera into the cornea account for most of the variations in the gonioscopic position of the trephine opening. Table I shows the relationship between position and result of 200 trephine operations.

TABLE I

Percentage of Successes	Positions		
	Anterior	Intermediate	Posterior
	24	72	24

Upon gonioscopic examination most trephine canals prove to have some "contents," that is, iris, ciliary processes, lens, vitreous, or a characteristic white, lacy tissue which is the product of local tissue proliferation. A small amount of content is usually insignificant and harmless. Obstruction by displaced tissues of most of the inner aperture is an unfavorable sign but not always associated with failure of the operation. Such voluminous contents were present in 50% of the failures, but in only 18% of the successes in a series of 200 operations performed at the Illinois Eye and Ear Infirmary. The large tissue inclusions in the trephine hole occurred most commonly in the most posterior positions of the latter. Postoperative absence of the anterior chamber for more than 12 days seemed to favor the occurrence of large tissue incarcerations into trephine canals.

Such large tissue incarcerations are present in about half of the unsuccessful trephine operations. The gonioscopic findings in the other half are either those of a completely filled in trephine canal or those of a dome-shaped structure; that is, closure of the outer portion of the

trephine canal. Extremely anterior position and ragged edges of the canal were the probable causes of the failure of some of these operations. In others gonioscopy revealed no clue concerning the mechanism of the obstruction.

In most of the cases in which the trephine operation has normalized the ocular tension, the gonioscopic and external biomicroscopic findings clearly and very impressively indicate the presence of a fistula that connects the anterior chamber with the conjunctival sac, with a few fine membranes interposed to slow down the flow within the fistula.

The Iris Inclusion Operation

Here the gonioscopic findings are not very revealing. Success of the operation does not seem to be related to the position or direction of the incision, or to the position of the iris within the incision (inverted or everted). In most of the successful operations and in a fair percentage of the unsuccessful operations, the inner aperture of the incision gapes permanently. The iris inclusion probably acts like a door jamb. Failure of the operation is usually due to factors which cannot be recognized by gonioscopy.

Cyclodialysis

There is close correlation between success of the operation and the persistence of a supraciliary cleft in the area of the cyclodialysis. Favorable factors are roominess of the angle and absence of firm peripheral anterior synechias. The spectacle of slow late closure of the pocket can be watched closely, but these observations have so far not revealed any clues concerning the mechanism and the prevention of these late closures. In the successful operation the supraciliary cleft apparently becomes a new channel of outflow.

Iridectomy

In the cases in which an iridectomy has successfully terminated one and prevented further attacks of narrow angle glaucoma (the latter without the aid of miotics), the gonioscope reveals at least a partially open, normal angle, and a direct communication between the chambers at or near the root of the iris. Provocative tests such as the darkroom or the mydriasis test, which before the iridectomy caused an abrupt rise on ocular tensions, are now ineffective. In all probability the successfully iridectomized eye "gets along" by means of its normal outflow channels.

Failure of the operation is associated with a completely, or almost completely, closed chamber angle or with gonioscopic indications of wide angle glaucoma.—*Author's abstract.*

Lacrimal Apparatus

Ectopic Lacrimal Puncta. A Congenital Anomaly Associated with Absence of the Lacrimal Papillae and Causing Chronic Tearing. *Arthur H. Keeney, Louisville, Ky.* Am. J. Ophthalmol. 32: 1586–88, Nov. 1949.

In cases of chronic tearing attention is directed to malposition of the puncta, not secondary to ectropion. The embryology of the canaliculi and papilla is reviewed and the more complex development of the lower segment is associated with the greater incidence of anomalies in this division. Usually described anomalies of the puncta are: (a) total absence, (b) dimplelike imperforation, (c) atresia, (d) occlusion with epithelial plugs, (e) elongation, (f) shallow gutter openings in the canaliculi, and (g) accessory puncta. To these should be added ectopic puncta.

Eight cases are collected from the literature and an additional (bilateral) case is reported in a 23-year-old male complaining of watery eyes since birth. The puncta in this case are on the lacrimal rather than the ciliary portion of the lid border, and the inferior openings are directed superiorly so as to be out of communication with the lacrimal lake. No papilla can be seen. Eye and nose exams including lipoidal X-rays of the lacrimal passages are otherwise negative. This defect is apparently due to an aberration in the developmental course of the canalicular buds at the 34 to 35 mm. stage, whereby they open through the lacrimal portion of the lid margin instead of continuing laterally to the normal site. 7 references, 1 figure.—*Author's abstract.*

Eyelids

A Simple Intervention for Relief of Spastic Entropion. *(Ueber einen einfachen Eingriff zur Behebung des Entropium spasticum.)Otto Schöpfer.* Klin. Mbl. Augen. 115: 40–42, Heft 1. 1949.

The large number of treatments suggested for the relief of spastic entropion indicates that satisfactory results are few. The good results obtained by enucleation of the tarsus in entropion due to trachomatous cicatrization suggested a procedure that might prove useful in cases of spastic entropion. Following instillation of 2 to 3 drops of 1% pantocain into the conjunctival sac, 1.5 to 2 cc. of 2% novocaine are injected into the transitional folds and subcutaneously in the vicinity of the lid margin. The eyelid is seized with a strong anatomic forceps at a depth of about 1 cm. in the center of the margin and perpendicular to it and is everted. With the lid thus fixed, double sutures are inserted 1 cm. from the margin of the lid and at a distance of 5 mm. from each other, to be brought out obliquely through the eyelid at the level of the eyelashes. A second and third suture are placed medial

and lateral to these. The sutures are then pulled taut and tied over tiny gauze pellets to prevent cutting through the lid. Slight exaggeration can be easily compensated. Should the suture be spontaneously expelled, no harm results, since the scar exerts the needed traction. The results of this intervention are due to displacement of the falsely placed orbicularis muscle bundle by traction of the suture or scar fixation. 10 references. 5 figures.

On Genesis and Operation of the Cicatricial (Trachomatous) Entropion of the Upper Lid. *A. Kettesy, Debrecen.* Brit. J. Ophthal. 32: 419-423, July 1948.

It has been observed that trachomatous entropion begins with cicatricial shrinking of the tarsal conjunctiva with drawing down of the fornix. The Meibomian ducts and later the skin, are drawn in contact with the eye. The intermarginal surface of the tarsus is rounded off into the conjunctival tarsal surface and thus a repair should involve remaking the posterior half of the intermarginal surface.

A new operation is described in which an incision is made through the tarsus in the scar line of the sulcus subtarsalis. Three mattress sutures are inserted in the conjunctival margin of the section. The marginal tarsal strip is freed from the skin, rotated and fixed by the mattress sutures placed perpendicularly through the tarsal strip. The suture ends are then drawn up and fixed to skin above to form an early overcorrection. The author has used this operation with success since 1930. 5 figures.

External Ocular Malignancy. *Brendan D. Leahey.* Presented at the American Academy of Ophthalmology and Otolaryngology, 1949.

Melanomas of the conjunctiva and skin are very common. They may appear at birth or later. They may remain unchanged through life or they may become the source of malignant tumors. When incompletely removed, they may be followed by wide spread dissemination through blood vessels and at times through lymphatics.

The congenital ones are called nevi. They are usually not seen at birth because they are small and more or less flesh colored. They grow in early years and become pigmented between the age of four and fourteen. There may be a rapid change in pigment content in adult life which both patient and doctor may mistake for growth. A nevus may develop into a malignant tumor, but this is very rare. The process of oxidation of premelanin into melanin is reversible; it may get darker and then lighter again, or it may lose all of its pigment.

It is often impossible to tell which melanoma may become malignant. Reese has given us an excellent description of pre-cancerous melanosis. "At the average age of from forty to fifty years, there may appear

on the skin of the lids, or the conjunctiva, or both, a non-elevated, diffuse, mottled, and more or less granular appearing pigmentation which may remain with little or no change in a pre-cancerous state for from five to twenty years before becoming malignant." In the pre-cancerous stage, the melanoma is radio-sensitive, but in the malignant stage, it becomes radio-resistant. Sixty-five per cent of all malignant melanomas develop from previous benign melanomas. On the skin ones, most significant symptoms are: increased size, darkening of pigmentation, and occasionally ulceration or bleeding. The margins are usually indefinite, and there may be fingers of brownish-black pigment extending from the tumors; and as it grows, well delineated, slightly elevated satellite nodules may be observed.

A malignant melanoma of the eye may have a very characteristic evolution in that many years may elapse before the tumor again becomes apparent, nearly always in the liver, often in the brain.

Treatment: All melanomas of the conjunctiva should be treated when small and before they are malignant. In general, complete excision with pathological examination is to be preferred. Radiation is not reliable. Since some benign melanomas become malignant after injuries from chemicals, insufficient radiation, or other trauma, biopsies of melanomas are distinctly contra-indicated.

When the melanomas are on the iris, important signs of malignancy are increase in size, secondary glaucoma, low grade irido-cyclitis with pigmented precipitates on the posterior cornea and impaired iris motility near the melanoma.

Lymphoma: In the origin of lymphosarcoma, two specific cells participate, giving rise to two specific forms of tumor. These cells are 1) the reticulum cell of the germ centers of follicles and lymph cords and 2) the lymphocytes. Two types thus arise, the reticulum cell sarcoma, or large round cell sarcoma and the malignant lympho-cytoma. Since tumors of lymphoid tissue respond to radiation better than any other group, radiation is usually the treatment of choice. On one extremely large malignant lymphoma a single dose of 800 r appeared to give complete resolution of the lesion, but this should be repeated again later.

Angioma: When the vascular channels are widely dilated and the connective tissue walls are thin, the angioma is designated as cavernous. The process is frequently seen in nearly all tissue and organs, but is especially common in the anterior orbit. These may increase in size and cause pressure and even erosion of bone. Growth is stimulated during menstrual periods and pregnancy. Rare cases may ultimately become malignant by changing to a hemangio-endothelioma. Surgery is often difficult in these deep-seated cavernous angiomas. Radiation is effective, but has many serious disadvantages. On several of these we have had excellent results with four to six injections of 5% sodium

Morrhuate (1 cc. each followed by pressure bandage) given two months apart.

Xeroderma Pigmentosum: may be an aggravated form and sequel to common freckles. It is more common in sunny climates, and starts early in life. It is marked by over-growth of pigmented epithelium scaling, atrophy of the affected skin, and formation of multiple little skin tumors, especially epitheliomas, over the exposed areas of the body.

Epidermoid Carcinoma: Histologically, epidermoid carcinoma is divided into two main varieties, squamous cell carcinoma and basal cell carcinoma. Both types occur sometimes in the same lesion. The squamous cell carcinoma may pass through a preliminary papillomatous stage or it may be flat, depressed, indurated, and infiltrating from an early period. Later the lesion gradually extends in the form of a broad ulcer with raised nodular indurated edges and granulomatous base. It may spead along blood vessels and nerves and invade lymphatics to metastasize to lymph nodes. It is a more rapid and destructive lesion than the basal cell type.

The typical basal cell, or rodent ulcer, starts as a flat papule or smooth wart which long remains without marked change. The early lesion may appear as a broad flat elevation or as multiple small thickenings which coalesce. After months or possibly several years superficial ulceration occurs. The edges are raised, nodular, indurated, pearly and constantly hyperemic. In advanced stages, the ulcer may become very wide involving much of the face. The eye, ear, and nose are often completely destroyed. The periosteum is invaded with destruction of bone and penetration of bony sinuses. The lymph nodes are seldom invaded in basal cell even after years of growth and spread is direct with little danger of metastasis.

Incidentally, Ewing says squamous cell carcinoma is considered almost exclusively the result of chronic irritation of the skin such as exposure to sunlight or x-rays, or a sequel to friction, chronic ulceration, eczema or burns.

In each of these cases, we are called upon to decide whether to use radiation or surgery. Basal cells are quite radio-sensitive but the squamous cell carcinoma is more radio-resistant and must be treated early. When the tumor is on loose skin, not involving the lid margin, excision is the quickest and best way and leaves less scar afterward. On the side of the nose the skin is thick and stiff and difficult to close over. The white radium scar is not too noticeable here, so radiation is more frequently used. Epithelioma of the lid margin requires a more radical operation, but if well done, will sometimes leave less scar than radiation. Unfortunately, to be effective, enough radiation is usually given to cause a secondary reaction and the scar formation may leave lid deformity or entropion. Also we occasionally get telangiectasis. On very old people we lean toward radiation on lid margin cases, and

in others we lean more heavily toward surgery, but decide each case individually. Another deciding factor is the size of a lid margin lesion. On a small defect, radiation rather than a radical resection and plastic is used. If the lid margin lesion is very large, however, radiation is bound to leave extensive deformity. Here, we prefer radical surgery with plastic repair for best cosmetic results.

If radiation is chosen, the dosage must be adequate, as the inadequate dosage makes the tumors more resistant. The most stubborn cases are those who have received repeated small doses. The tendency now is toward one or two large treatments.

When surgery is used and the tumor is of fairly malignant type, radiation may be used later around the area of the incision and over the regional glands. It can be used immediately on the glands and after two to four weeks on the area of the incision.

Carcinoma of the Eyelids. *Edith M. Parkhill, Mayo Clinic.* Presented at the American Academy of Ophthalmology and Otolaryngology, 1949.

The commonest malignant tumors of the eyelids and canthi are carcinomas, and malignant tumors of other types are found only rarely in this location. Practically all of the carcinomas are either basal-cell carcinomas or squamous-cell epitheliomas; of these two groups approximately 85% are basal-cell carcinomas and 15% squamous-cell epitheliomas. Included among the basal-cell carcinomas are those with areas of squamous-cell differentiation (the basosquamous epithelioma of Montgomery). Melano-epitheliomas make up about 2% of the malignant epithelial tumors of the eyelids and canthi. An adenocarcinoma is encountered in these locations in about 1% of total instances of malignant lesions of epithelial origin.

The incidence according to location varies: 54% of the malignant tumors of the eyelids are found on the lower lid, 12.5% on the upper lid, 24.5% at the inner canthus, and 9% at the outer canthus (Birge). On the lower lid basal-cell carcinomas exceed in number the squamous-cell carcinomas by a ratio of almost 6 to 1; on the upper lid the basal-cell carcinomas are relatively fewer and predominate only by a ratio of about 2 to 1.

Many factors have been suggested as causative in the pathogenesis of these tumors. Prolonged exposure to sunlight plays a part.

According to Broders the incidence of carcinomas of eyelids and conjunctiva is 3% of the total incidence of epitheliomas. Basal-cell carcinomas comprise 5% of skin cancers (Geschichter and Koehler).

Birge, in a study of a series of cases of epithelioma of the eyelids,

canthi, cornea and conjunctiva, which had been classified as to type, found that of patients with epithelioma of the upper lid, 40% of those with squamous-cell epithelioma and 14% of those with basal-cell epithelioma were known to have died of "eye cancer" within the traced period, whereas of those with epithelioma of the lower lid, 15% of those with squamous-cell epithelioma and 8.5% of those with basal-cell epithelioma were found to have died of "eye cancer" within the same period. During the same period the mortality for patients with tumors classified as squamous-cell epithelioma as well as for those with basal-cell epithelioma of the outer canthus was nil, whereas among the patients with epitheliomas of the inner canthus, 33% of those with squamous-cell epithelioma and 16% of those with basal-cell epithelioma were found to have died of "eye cancer."

Results varied also according to degree of malignancy. For the patients who had squamous-cell epithelioma, grade 1 (Broders' method) the five-year survival rate was 100%; among those who had squamous-cell epithelioma, grade 2, the five-year survival rate was 82%; for those who had squamous-cell epitheliomas, grade 3, the five-year survival rate was 58.3%, and of those with squamous-cell epitheliomas, grade 4, all of the patients traced for the five-year period were known to be dead.

Grossly the basal-cell epithelioma can usually be recognized as a smooth or scaly, slightly elevated papule, often with central ulceration rimmed by a waxy, smooth, rolled border.

Microscopically the basal-cell epithelioma appears as nests of closely packed uniform cells with a peripheral layer of columnar cells arranged parallel to each other and perpendicular to the so-called basement membrane. Variations are the pigmented basal-cell epithelioma; the morphea type (dense hyaline stroma between small nests and cords of tumor cells); the basosquamous type previously mentioned; the cystic glandular type; and the comedo type with necrotic centers to cell nests.

The squamous-cell epithelioma generally appears as a scaly, indurated, crusted lesion, and may show ulceration or hornification. It is made up of hyperchromatic epidermoid cells showing a greater or less degree of anaplasia, usually arranged in strands or irregular cell nests with loss of polarity of the peripheral layer of cells. There may be prickle-cell formation; more frequently some of the cells are keratinized. Squamous-cell carcinomas of the eyelids generally are not highly malignant.

The rare adenocarcinomas are usually of a low degree of malignancy. They vary in histologic type; some may be of sweat gland or sebaceous gland type; rarely a mucus-producing adenocarcinoma is seen. —*Author's abstract.*

Differential Diagnosis and Treatment of Ptosis. *Paul L. Cusick, Detroit, Mich.* Presented at the American Academy of Ophthalmology and Otolaryngology, 1949.

A simple classification of cases of blepharoptosis may be made as follows: 1) Congenital. 2) Paralytic. 3) Traumatic. 4) Pathophysiologic. 5) Pseudoptosis.

The congenital form is the most common, and in cases where there is some action of the levator a resection of this muscle gives the most satisfactory cosmetic and physiologic results. However, with this procedure, as with all others, some exposure of the globe may be present during sleep. This should be discussed with the patient or parents before surgery. This is usually minimal, however, with the Blaskoviez technic or a modification of it. The levator muscle is similar to the rectus muscle in that after resection it does not relax to the same point as previous to surgery.

In doing a Blaskoviez operation or a modification of it, the missteps in technic which cause difficulty are mainly as follows:

1) In reflecting the conjunctiva, more than just conjunctiva is reflected. Part of the levator aponeurosis may be reflected inadvertently. It should be remembered that the conjunctiva is essentially as thin in this region as on the globe itself.

2) Over correction. This is rare but if it occurs the remaining levator may be recessed according to Goldstein's technique and a satisfactory result obtained.

3) In reflecting the levator, the orbital septum may be incised and reflected also. This results in an impasse when an attempt is made to free the supposed anterior surface of the levator, the orbital septum being reflected forward toward the superior orbital margin. Then it may be perforated and resected with the levator resulting in a splinted lid and poor physiologic result.

4) Not enough levator is resected, resulting in undercorrection. The approach in these cases must be similar to that in marked divergent strabismus where we resect all the tendon possible.

5) Failure to sever the lateral extensions of the aponeurosis of the levator, in the larger amounts of ptosis especially. In children with smaller amounts this is not usually necessary.

6) Resecting a disproportionate amount of the central portion of the levator, resulting in a "bowed" lid.

No operation requires more attention to detail, but the results justify the trouble.

The complication most feared in any ptosis operation is ulcerative keratitis. The author has observed three cases and has been told of a number of others. Nearly all have been associated with the Motais type of operation and some have resulted in lawsuits. In direct gaze the

cosmetic results from this type of operation are usually good. Physiologically, however, they are not so good. Both elevation and depression of the eyeball are interfered with and lid-lag is always present in downward gaze. The author has discontinued this type of operation. It is felt that a sling-type frontalis operation is preferable when a resection of the levator may not be done.

In all ptosis operations both an intermarginal and a frost suture are used to protect the cornea.

A resection of the levator for ptosis associated with anophthalmos has not been satisfactory in the author's hands. The direction of pull of the levator is not normal and the cosmetic result may not be good. A sling-type operation has been most satisfactory in these cases. The latter is also used for simple complete paralytic ptosis.

In traumatic ptosis the approach will depend on the type of injury. If it is due to the lack of repair of the levator at the time of the injury, the levator may be resected as advocated by Spaeth. However, if, as is usual, the eyeball is missing, a sling-type operation will probably be more satisfactory.

A motion picture was used to demonstrate steps and missteps in the Blaskovicz procedure and to illustrate a number of different types of ptosis.—*Author's abstract.*

Orbit

Hydatid of the Orbit. *Sir Henry Holland, India.* Brit. J. Ophthal. 32: 395–396, July 1948.

Little has been writen about orbital cysts from entozoa echinococcus, so-called hydatid tumors. The author's experience with eight cases indicates that the cysts generally appear on the nasal side and upper fornix. Pain is seldom a feature of the disease. Proptosis varies with the duration of the disease and may progress to such an extent that the patient cannot close the eye. The diagnosis can be established by aspiration of the cyst and microscopic examination of the contents.

Treatment consists of removal of the cyst which may be difficult because of the friability of the cyst wall. The vision of two patients was unimpaired by treatment; the six other patients could not be followed adequately.

Orbital Lesions: Differential Diagnosis and Treatment. *Jerome W. Bettman, San Francisco, Calif.* Presented at the American Academy of Ophthalmology and Otolaryngology, 1949.

The cardinal sign of orbital lesions is exophthalmos. The subject of exophthalmos is difficult because of the numerous possible causes. If the uncommon causes are eliminated there remains a workable group which accounts for the vast majority of cases and permits logical con-

sideration of the clinical diagnosis and approach to treatment. No attempt is made to discuss all causes of exophthalmos. There are five principal causes of exophthalmos: tumors, systemic disorders, inflammatory conditions, trauma, and congenital abnormalities.

Tumors are usually unilateral and so is the exophthalmos produced by them; therefore, unilateral exophthalmos suggests tumor. Tumors may be primary in the orbit, secondarily invasive from neighboring structures, or metastatic.

The commoner orbital tumors are: 1) Hemangioma: Congenital origin, usually apparent in the first three decades. Two noteworthy characteristics—does not affect the motility of the eye, and tends to vary in size. Compressible and noncompressible hemangiomas. Compressible ones are likely to be the cavernous type, diffuse, difficult to shell out surgically, and X-ray therapy is the treatment of choice. Noncompressible hemangiomas are cellular, tend to be encapsulated, do not expand, shell out readily, and, therefore, may be locally excised.

2) Pseudotumor: Granuloma of unknown etiology. Characteristic findings which are often absent—edema and redness of the lids weeks or months before exophthalmos in approximately one-half the cases; some pain, bilateral in one-third; second eye involved at the same time or up to nine months later. Bilaterality with prodromal edema may differentiate from tumor. Age of onset, average 45 years, somewhat older than for most tumors. Spontaneous regression; therefore, no treatment if one is certain of the diagnosis.

3) Meningioma: Hyperostosis characteristic, but not pathognomonic. Only meningiomas of the sphenoidal ridge need be considered. Those arising in the middle third of the ridge result in extensive hyperostosis and exophthalmos without subjective symptoms until later. Meningiomas of the inner third produce early optic atrophy, later papilledema of the contralateral side, maybe minimal X-ray changes, variable exophthalmos. Meningiomas of the outer third produce exophthalmos in the homolateral eye, palpable swelling on the same side, slowly increasing impairment of vision in the order mentioned; also thickening and erosion of bone. Treatment by neurosurgeon.

4) Dermoids: Congenital, usually increase in size at puberty. Superficial dermoids may extend backwards and produce proptosis. May be deep and produce optic atrophy or papilledema; may be bone defect demonstrated by X-ray. Treatment by simple excision.

5) Sarcoma: Commonest cause of noninflammatory unilateral exophthalmos in children under 12. If rapidly progressive and with bone destruction, strong presumptive evidence. Exenterate, but usually fatal. Another group in older people. Lymphosarcoma different, usually in older people, usually isolated lesion at first responds well to X-ray.

6) Neurogenic tumors: a) Neurofibromas of von Recklinghausen:

Congenital origin, associated with other signs. Tumor is soft with knotty cords like "bag of worms." May be associated with defects in orbital bones. Tends to recur after excision. Very bloody. b) Glioma: Congenital usually before fifth year. Unilateral proptosis with loss of vision, but good motility in a child.

7) Mixed tumor of lacrimal gland: Half of the cases associated with exophthalmos. Marked tendency to recurrence, local invasion, and progressive malignancy. Excise widely.

Tumors from neighboring structures are: 1) Benign: a) Mucocele: Usually frontal or ethmoidal. Diagnose by X-ray. Don't approach from orbit. b) Osteoma: Diagnose by X-ray. 2) Malignant: a) Nasopharyngeal tumors: Always have nasopharynx examined. b) Malignancies from sinuses: Usually maxillary. Simulate cellulitis, as do nasopharyngeal tumors. c) Basal cell carcinomas from lids: Invade underlying tissue, go retrobulbar, produce inflammatory reaction. d) Metastatic malignancies.

Systemic Lesions

Thyroid group: Thyrotrophic excess, edema of lids and conjunctiva, real proptosis, extraocular muscle paralysis, may appear to be unilateral. Dangerous to vision and to the eye. Avoid by care in selection for thyroidectomy. If proptosis, treat promptly with thyroid if possible; do decompression early; best done temporally. Thyrotoxic excess produces appearance of proptosis, but not real.

Xanthomatosis: A disorder of lipoid metabolism in which cholesterol is deposited to form tumor masses. Two forms considered:

1) Schueller-Christian disease in young children characterized by exophthalmos, diabetes insipidus and bone defects in skull. 2) Localized tumors in adults especially arising from frontal bone and going into orbit.

Paget's disease and leontiasis ossea.

Traumatic Lesions

1) Arterio-venous fistula: Pulsating exophthalmos. 2) Retro-orbital hemorrhage.

Congenital Lesions

1) Intracranial aneurysms. 2) Craniostenosis: Tower skull or oxycephaly. 3) Unilateral myopia of high degree.

Inflammatory Lesions

Orbital abscess: Orbital cellulitis. Cavernous sinus thrombosis.

Commoner Causes of Bilateral Exophthalmos

1) Thyroid disorders. 2) Craniostenosis. 3) Cavernous sinus thrombosis—not to be confused with tumors. 4) Neurofibromatosis—

previously discussed. 5) Hypothyroidism—may produce exophthalmos—thyrothropic hormone? 6) Mikulicz's disease—nodules on lids, responds to X-ray, unlike thyroid. 7) Angioneurotic edema—unusual, but may last months or more.

Treatment

In handling a case in which the diagnosis is not apparent, first use medical, ENT, neurological and roentgenological consultations freely. If tumor in anterior orbit, remove frontally; if in posterior orbit, have removed transcranially. If presumption of tumor and threatened visual loss, explore orbit, but be prepared to complete operation. If vision not threatened and diagnosis in doubt, remember it is no emergency; give X-ray therapy and await developments. Don't open an orbit simply for biopsy.

Allergy

Contact Allergy to Plastic Artificial Eyes. *J. MacIvor.* Canad. Med. Ass. J. 62: 164–6, Feb. 1950.

Eight cases of contact allergy appeared in a hundred patients who were fitted with plastic prostheses. There was a burning sensation, the socket became red and edematous, the symptoms disappeared when the plastic was removed. A patch test in the forearm with the plastic was positive in two of the eight cases, negative in the remaining six cases. In all cases there was an allergic response to a button of plastic in the socket. Examination of the plastic eye showed no cracks or roughness which could have caused the symptoms. Bacteriological studies did not reveal organisms which could have been pathogenic. 4 references.

One case has been seen that was sensitive to the plastic of contact glass.—Editor.

Pharmacology, Toxicology and Therapeutics

Acute Methyl Alcohol Poisoning. (*Zur akuten Methylalkoholvergiftung.*) *Heinz Geserick.* Klin. Monatsbl. f. Augenh. 114: 502–11, 1949.

Three cases of methyl alcohol intoxication are reported in detail. The ophthalmoscopic findings and changes observed in the visual field seemed to throw new light on this condition. The findings indicate that in acute methyl alcohol poisoning the changes in the arterial retinal vessels and particularly in their vicinity play a more important role than hitherto suspected. It is even possible that the primary lesion following acute methyl alcohol intoxication may be looked for in the arteries and nerve tissues in their vicinity, thus suggesting that methyl alcohol

is primarily angiotropic and only secondarily a neurotropic poison. Further investigation is urged as of possible interest for the treatment of acute methyl alcohol poisoning. In industrial medicine and chemical factories cases of beginning methyl alcohol intoxication have hitherto been classified as asymptomatic.

Retrobulbar Alcohol Injections. Relief of Ocular Pain in Eyes With and Without Vision. *A. Edward Maumenee, San Francisco, Calif.* Am. J. Ophthalmol. 32: 1502–08, Nov. 1949.

Retrobulbar injections of alcohol produced a dramatic relief of pain for at least one to three months in 35 patients with blind eyes and in 15 patients with partial vision. Transient ocular palsies occurred but no permanent complication, such as injuries to the optic nerve, extraocular palsies, neuro-paralytic keratitis or sloughing of the cornea were noted.

The technique used for retrobulbar alcohol injection was similar to that for retrobulbar injections at the time of intraocular operative procedures. A 3.5 cm. 22-gauge needle was inserted into the lateral third of the lower lid just above the rim of the orbit. The needle was passed through Tenon's capsule and into the muscle cone as close to the back of the globe as possible. If the tip of the needle was placed near the apex of the orbit, a higher percentage of temporary extraocular palsies occurred than if the needle was held close to the posterior surface of the globe. One cc. of 2% novocaine was injected, the syringe was removed leaving the needle in place, and after three to five minutes, the corneal sensitivity and extraocular muscles were tested to be sure the needle was in the proper position. A syringe containing 1 cc. of ethyl alcohol was attached to the needle and the solution injected into the orbit. In a few injections, 45% alcohol was used, but in most instances 95% alcohol was used because it was thought to be more effective in relieving pain and it did not increase the complications from the procedure.

A retrobulbar alcohol injection of 1 cc. of 95% ethyl alcohol was done on 41 eyes with less than 10/200 vision. In 6 patients, the pain was not alleviated. In 35 patients the pain was alleviated for at least one month; 15 of these eyes were removed after alcohol injections. In 13 instances the optic nerve was atrophic because of advanced glaucomatous changes, but in 2 cases the optic nerves were normal. In no instance was there any evidence of cellular infiltration in the optic nerve sheath to indicate that the alcohol had caused any undue irritation of the optic nerve.

Retrobulbar alcohol injections were made in 15 patients whose final visual acuity ranged between 10/200 and 20/15. A table is given which lists the type of ocular lesions, the duration of the pain before alcohol injection, the amount of alcohol injected, the visual acuity before and after injection and duration of the follow-up. In 3 of these

15 cases, 1 cc. of 45% alcohol was used and in 11 of the cases 1 cc. of 95% alcohol was used. In 1 case, it was necessary to inject 2 cc. of 95% alcohol before the pain could be relieved. There was not a single instance in this series of patients in which either a loss of central or peripheral visual acuity resulted from the retrobulbar alcohol injections. 11 references. 3 tables.

Giant Calcio-Osseous Hydro-Hematonephrosis. Angiospastic Retinitis or Exogenous Toxic (Sulfamide) Injury of the Eyegrounds. (*Riesige kalkig-knocherne Hydro-Hämatonephrose. Retinitis angiospastica oder exogen-toxischer (Sulfonamid-)Schaden des Augenhintergrundes.*) *Gottfried Wilhelm Günther and Johann Maurath, Baden-Baden.* Arch. f. klin. Chir. 262: 423-37, Heft 5/6, 1949.

A man of 38 years was operated on for a tumor in the abdomen causing vomiting and nausea. A giant left hydro-hematonephrosis containing 7 liters of thin bloody chocolate colored fluid was removed. The wall of the markedly dilated pelvic calyx was partly calcified and ossified, so that clinically and roentgenologically a teratoma had been expected. The etiology and pathogenesis of calcio-osseous hydro-hematonephrosis are discussed. There had been a marked hypertension which subsided following removal of the giant hemato-hydronephrosis. Finally, during the course of a febrile pyelonephritis, the patient was placed on a salt-free diet and Neo-Uliron was administered. During this period the patient lost his eyesight. The changes in the eyegrounds suggested angiospastic retinitis but the blood pressure had diminished. Together with the blindness he developed a severe anemia and later bilateral paralysis of the peroneal nerve. All of these changes which retrogressed almost completely, must be attributed to the association of a possible individual hypersensitivity or favored by the hydronephrotic solitary kidney, to some exogenous cause, *i.e.* the Neo-Uliron treatment. The problematic origin of the ocular changes is discussed. Hitherto changes in the eyegrounds have not been described as due to the sulfamides, but the nature of the lesions corresponded to changes in other organs produced by these drugs.

The patient was discharged 3 weeks after operation in good general condition. Fourteen days later he was placed on the salt-free diet and 48 tablets of Neo-Uliron were administered in 4 days for the first sulfamide shock and followed after an interval of 5 days by 20 more tablets. Thirteen days after he lost his vision he was able to read again. Examination revealed retinitis hemorrhagica in both eyes, which indicates a poor prognosis. However, within the following months the ocular changes and the paralysis retrogressed.

A Case of Pyocyaneus Ring Abscess of the Cornea Treated with Streptomycin. *J. Maschler, Haifa, Israel.* Brit. J. Ophthal. 32: 426–428, July 1948.

A ring abscess of the cornea developed following a corneal injury. On the fifth day following trauma the whole cornea was involved except for a narrow peripheral rim and slightly transparent central portion. A hypopyon filled a quarter of the anterior chamber. A diagnosis of *B. pyocyaneus* was established by culture.

Fifty thousand units of penicillin were injected subconjunctivally, the hypopyon was washed out and penicillin was injected into the anterior chamber. On the sixth day the patient received streptomycin topically every hour, intramuscularly every three hours, subconjunctivally daily and one injection of 2,500 units in the anterior chamber. He received 6 Gm. of sulfadiazine daily.

On the eighth day a broad Saemisch's section was performed and the corneal infiltration rapidly declined. A secondary glaucoma during the healing process was successfully controlled by iridectomy. The patient's vision was finger counting.

The organism was found sensitive to 500 units of streptomycin bacteriologically. 5 references.

A Case of Keratomalacia Cured by Penicillin and Vitamin A. *Kamel Rizk, El-Minia, Egypt.* Brit. J. Ophthal. 32: 416–419, July 1949.

A 2-year-old male infant was seen with generalized emaciation. Both corneae were becoming opaque, insensitive, dry, and ulcerated without inflammatory reaction and developing ectasias.

Penicillin was administered as drops in 2,500 u./cc. concentration supplemented by intramuscular injections. Improvement was marked without vitamin A but local and systemic vitamin A was later given. Atropine was administered locally.

In five weeks all ectasia had disappeared leaving a dense central leukoma in an otherwise clear cornea.

Subconjunctival injections of penicillin combined with local and general vitamin A therapy are proposed as the best method of treatment. 4 references.

Industrial Ophthalmology

Visual Testing Technics. *Henry A. Imus, Washington, D. C.* Presented at the American Academy of Ophthalmology and Otolaryngology, 1949.

Until recently the function of the ophthalmologist in industry was commonly limited to giving first aid to injuries. But, today, his services have expanded to consideration of the selection and classification of personnel for job placement. Better placement in jobs has resulted in

greater satisfaction, increased earning power, as well as advancement and promotion for the worker.

Such regulations have reduced wastage, and aided early detection of abnormal ocular or visual conditions. To aid the ophthalmologist in his attention to the increased number of workers and in his maintenance of a regular routine of examination, several instruments have been invented with which to obtain responses to tests of sight and ocular muscle powers.

Fact-Finding Methods in Industrial Vision. *N. C. Kephart, Purdue University, Lafayette, Ind.* Presented at the American Academy of Ophthalmology and Otolaryngology, 1949.

Kephart offered a course on "Fact-Finding Methods in Industrial Vision," based upon his experience at the "Occupational Research Center" at Purdue University at Lafayette, Indiana. A psychologist, he has been made aware of the great difference between workmen employed on identical jobs. His results are obtained not by a psychological preconception of the powers of an individual, but by a painstaking analysis of findings investigated by the establishment of a definite mode of examination.

The results have been tabulated and expressed graphically, showing scores manifested by relations between vision and job efficiency; the lower the response to vision-tests, the less the efficiency, and the higher degree of sight, the more efficient the products. The scores can be evaluated and the results submitted to both the worker and his employer.

In the course of time, standards of sight and work-output have been adopted and accepted and demanded of workers seeking employment, and the results manifested by large numbers of workmen have justified their adoption by employers who have been guided thereby, in their decision in assigning the worker to his job.

Eyes and Industry. *Hedwig S. Kuhn, Hammond, Ind.* Presented at the American Academy of Ophthalomolgy and Otolaryngology, 1949.

As in the case presented by Imms, Kuhn too insists that that service has a far-reaching function to include the estimation of the visual powers of the job-applicant to fit him for employment and to be concerned with his efficiency as a craftsman.

Knowledge of eye hazards and means of protection should be known, well understood, and adequately protected from the demands which require that the attending officer should be competent to exercise the modern methods of first aid in accidents, and wise and effective counsel to the industrial worker and his employer.

The relation to compensation for injuries and the value of time-

losses demand knowledge and put the ophthalmologist in a position of responsibility not previously demanded or asserted.

As pointed out by this review years ago, the illumination of the workroom and the clearing of the atmosphere must be provided for adequately and with advice from illuminating engineers.

Miscellaneous

A Survey of Visual Complaints Related to Television. *Henry W. Hofstetter, Los Angeles.* Am. J. Optometr. & Arch. Am. Acad. Optometr. 26: 483–9, Nov. 1949.

The survey was designed to determine the prevalence of visual complaints in a large sample of optometric patients. Data were obtained from 292 clinical consultations with patients receiving routine optometric examinations at the clinic of the Los Angeles College of Optometry and at the private offices of fifteen optometrists in the Los Angeles area. The prevalence was evaluated in terms of certain non-optometric information such as age, sex, frequency of viewing, and the method of eliciting the complaint.

Patients with visual complaints readily identify their visual discomforts with television viewing. There is an absence of correlation of complaints with age. There appears to be no correlation of complaints with frequency or duration of viewing. The variety of complaints suggests that television viewing difficulties are not readily identified with a limited category of patients. Few patients, if any, were refusing to accept television on the basis of suspected eyestrain. Approximately one-third of the optometric patients in this study answered "yes" to the question, "Do your eyes bother you when you watch television?"—*Author's abstract.*

Although many people complain of visual symptoms, there is no scientific evidence that watching television can cause any organic eye changes.—Editor.

Associated Eye and Skin Manifestations of Systemic Disease. *Isadore Givner, New York, N. Y.* Presented at the American Academy of Ophthalmology and Otolaryngology, 1949.

The appearance of lesions in both the skin and eye as manifestations of general disease makes it important to be familiar with the expressions in both systems for the better recognition and handling of systemic disorders.

Phacomatoses (Van der Hoeve): a) Bourneville's disease: Sebaceous adenoma of the skin may occur with retinal cysts. The latter evolve into the classic mulberry appearance by the impregnation of calcium. b) Recklinghausen's disease: Molluscum fibrosum, moles, and café au

lait spots may occur with neuromatous changes in the ciliary nerves, sclera, cornea, uvea, and optic nerve. Bone involvement occurs in 70% of the cases, producing at times pulsating exophthalmos if the roof of the orbit is absent. e) Sturge-Weber's disease: Angiomatous changes can give rise to the simultaneous appearance of a nevus flammeus of the skin and glaucoma. The involved eye shows increased capillary permeability with the fluorescein test.

Amino Acid Deficiencies: Exfoliative dermatitis with associated hypoproteinemia may account for corneal dystrophy by a lack of valine. The lack of the latter experimentally produced edema and dystrophy of the epithelium in white rats, while the feeding of valine reversed the corneal pathology. Clinically the lesion is also reversed by high protein diets.

Acute Disseminated Lupus Erythematosus: The typical butterfly lesion on the face with associated kidney involvement may have striate exudates in the retina, the so-called cytoid bodies. The finding of the latter in a case with no hypertension or vessel attenuation, continued fever, and a rash brought on by exposure to sun or sulfonamides should make one suspicious of lupus erythematosus.

Boeck's Sarcoid: The superficial skin lesions are papular and nodular elevations, sharply outlined, dark red, and may be surrounded by adherent scales. The larger lesions are subcutaneous and nodular and have either faint or no surrounding erythema. The eye is a favorite site of this disease. The most frequent lesion is uveitis. The condition may take on the picture of Mikulicz's syndrome or Parinaud's oculoglandular syndrome.

Recurrent Aphthous Uveitis with Mucocutaneous Lesions: Buccal aphthae, recurrent aphthae on the genitalia, or ulcerations on the scrotum and balanopreputial groove, sometimes accompanied by nonspecific urethritis, may be associated with iritis, hypopyon, or only conjunctivitis. The possibility of a virus infection or a hypersensitiveness to foreign proteins has been considered in the etiology.

Vogt-Koyanagi Disease: Nontraumatic uveitis asociated with alopecia, poliosis, vitiligo, and dysacousia completes the picture. Cutaneous testing with pigment shows a positive reaction to that seen in sympathetic ophthalmia in about one-third of the cases.

Melanoma of the Skin with Intraocular and Orbital Metastases: Although most melanotic tumors of the eye are primary, the possibility should be borne in mind that they can be secondary, and careful search should be made for other primary sites and evidences of multiple metastases.

Brucellosis: Iritis, optic neuritis, chorolitis, uveitis, palsies of the ocular muscles and retinitis may occur with skin lesions of various types ranging from maculopapular eruptions to ulcerative dermatitis.

Miscellaneous: a) Pseudoxanthoma elasticum—angioid streaks of

the retina. b) German measles—congenital cataracts. c) Neurodermatitis—cataract. d) Pemphigus—subconjunctival fibrosis.

Further Contribution to the Study of Naso-Ciliary Neuritis: Genuine Interstitial Neuritis and Degenerative Parenchymatous Neuritis. (*Ulteriore contributo alla conoscenza della nevrite vera (interstiziale) e la nevrite degenerativa (parenchymatosa) o nevritide nasociliare*). *Vitanzelo Spadavecchia, Palermo.* Rivista oto-neuro-oftal. 24: 389–408, Sept.–Oct. 1949.

Following a review of the literature on naso-ciliary neuritis, the author describes two distinct forms of this disease, namely 1) genuine interstitial neuritis, which is well known clinically, and 2) a degenerative neuritis which is chiefly parenchymatous. This latter type is characterized principally by a nerve defect. The symptoms include corneoconjunctival hypoesthesia, hypoesthesia of the corresponding pituitary mucosa, neuroparalytic keratitis, incomplete Horner's syndrome, slight pain on pressure over the external nasal and nasolobular rami, with paresthesia in the distribution of the nerve. In this latter type there are no irritative attacks like those noted in the genuine interstitial neuritis. 5 references.

Results of Early Treatment of Congenital Anophthalmos. *William S. Kiskadden, Allyn J. McDowell and Ted Keiser, Los Angeles, Calif.* Plast. & Reconstruct. Surg. 4: 426–433, Sept. 1949.

The authors of the above article trace the embryological story back of this congenital deformity, and quote Mann, who has classified this deformity and summarized it into three categories: 1) Primary Anophthalmos, 2) Secondary Anophthalmos, and 3) Degenerative or Consecutive Anophthalmos. As Mann points out there are rarely other associated defects. The eyelids and remainder of the external anatomy of the eye are present and normal except for their diminished size. Because of the lack of a socket, the problem is one of increasing the existing socket, either surgically, or by other means, into one that is adequate to carry a satisfactory prosthesis. The experience in the past with surgical correction of these has been uniformly unsatisfactory. The authors present a case which shows the rather indifferent results obtained by surgical correction, and make a plea for the use of gradual dilatation by acrylic implants, starting as early as possible. Pictures of one case, and charts of the increased acrylic compounds used for dilatation are shown. The conclusions are that under gradual dilatation started preferably at an early age, a normal socket may be obtained into which can be fitted a quite satisfactory artificial eye. 5 figures. 14 references.—*Author's abstract.*

Ocular Defects in Still's Disease. *H. E. Hobbs.* Proc. Roy. Soc. Med. 42: 755–756, Sept. 1949.

A quiet chronic iritis is the usual, and may remain the only ocular lesion in juvenile rheumatoid arthritis; but an exudative form has been reported and synechia and cataract are known to occur. The case demonstrated was that of a girl of 10 in whom Still's disease had first been diagnosed at the age of 3; the general condition is described. Her eyes were first known to be affected at the age of 4 and at the present time her visual acuity is reduced to 6/36 in the right and 6/18 in the left eye with lenses. Both lenses show anterior subcapsular opacities with fine granular deposits from old iritis, although the pupils are mobile. Both corneae show opacities in the interpalpebral area which are seen under the slit lamp microscope to be localized to Bowman's membrane and the superficial corneal lamellae, and to have the faintly brownish turbidity with fenestrations characteristic of band-shaped opacities.

The author drew attention to the fact that ocular lesions tend to appear early in Still's disease, may, in fact, precede the joint symptoms, and should be regarded as a separate ocular response to the infective agent rather than a secondary "rheumatic" manifestation. 13 references.—*Author's abstract.*

The Rosacea Complex, Including Its Ocular Component. *Edward P. Cauley, and Arthur C. Curtis, Ann Arbor, Mich.* Am. Practitioner. 4: 119–23, Nov. 1949.

1) Rosacea is a facial disorder which early in its evolution is characterized by transient episodes of erythema and vasodilatation. After months or years, papules and pustules appear and the glandular orifices become conspicuous. An oily seborrhea involving the face is a frequent accompaniment. Subjective manifestations are minimal.

2) Rhinophyma develops in some cases of long-standing rosacea. It is characterized by an increase in size of the nose, often in regular fashion but occasionally in the form of irregular, lobulated masses, the color being dark red or dusky purple. The sebaceous orifices are widely dilated. Occasionally rhinophyma appears as the only evidence of rosacea, the remainder of the face being spared.

3) Ocular rosacea is common and may consist of blepharitis, conjunctivitis, keratitis, or a combination of all three. Rosacea keratitis is of greatest significance because it may result in corneal opacity.

4) Study of 350 case records of rosacea has shown:

   a. Most examples of the disease are encountered between the ages of 35 and 50.

   b. Oily seborrhea and seborrheic dermatitis are frequently coexistent with rosacea and there may be an underlying relationship between the latter two disorders.

c. Disturbances of the gastro-intestinal tract are far more common in patients who have rosacea than in those with other dermatoses.
d. Disturbances in gastric hydrochloric acid content are not a fundamental part of the rosacea complex.
e. An endocrine imbalance may play some role in the etiology of rosacea.
f. Rhinophyma appears to be more common in women than formerly believed.
g. Ocular rosacea is relatively common, easily overlooked when mild and a complication to be watched for in every case of rosacea.
h. In this series, rosacea-like tuberculid was the disease most often confused with rosacea. Microscopic examination of tissue was resorted to in several instances as an aid in differential diagnosis.

5) Simple topical measures plus small amounts of superficial x-ray are helpful in the treatment of rosacea, and a combination of this type of therapy, in addition to large doses of crude vitamin B complex by mouth and the relief of gastro-intestinal disturbances, are productive of definite improvement in most instances. Rhinophyma is best treated by removing redundant tissue with the electrical cutting current. Treatment of ocular rosacea, other than simple blepharitis, is best carried out in co-operation with an ophthalmologist who may utilize warm compresses, antiseptic eye drops, powdered calomel, beta radiation, surgical intervention or cauterization for ulcers, or other therapeutic measures as dictated by the individual case. 20 references.—*Author's abstract.*

Thyrotoxicosis, Exophthalmic Ophthalmoplegia, Myasthenia Gravis and Vitiligo. *Raymond Greene, London, Eng.* Proc. Royal Soc. Med. 42: 263–67, April 1949.

A woman of 27 was normal until in December 1946 she developed diplopia. In April 1947 she developed exophthalmos of the right eye and a patchy brown pigmentation of the skin of the whole body. Classical Graves' disease then rapidly developed. In June 1947 ptosis of the left eye was first noticed, and by August the picture of myasthenia gravis was complete. In October 1947 all eye movements were absent or weak and there was extreme muscular weakness of the arms and legs. Prostigmin caused a complete disappearance of the ptosis, the limb weakness and the external ophthalmoplegia, except for outward movements, which were unaffected. After thyroidectomy, the outward movements of the eyes improved, but other movements did not. Thymectomy was then performed. This was followed by a steady improvement in the function of the remaining muscles. Within a year she was symptom-free and the pigmentation had almost disappeared.—*Author's abstract.*

Classification of Ocular Diseases. (*Systematik der Augenkrankheiten.*) *Hermann Pressbergen, Stuttgart.* Klin. Monatsbl. f. Augenh. 115: 1–12, Heft 1, 1949.

In the place of classifications now based arbitrarily on etiologic, functional, anatomic or ophthalmologic factors, the author suggests that a classification be made of diseases of the eye according to the tissue involved and the pathologic process, so that the term employed may describe as nearly as possible the location and nature of the pathologic process. Many conditions present a combination of two or more simultaneous disease processes. Naming a disease for the man who discovered it should especially be avoided. The name of the disease should come first followed by the indication of the tissue involved. In this way the different types of corneal inflammation or of retinal degeneration would appear side by side for easy comparison and the whole nomenclature and classification of ocular diseases could be placed on a more uniform and scientific basis.

Changes in the Eye and Ocular Adnexa Due to Rabies and Its Treatment. (*Alteraciones que la rabia y el tratamiento provocan en el ojo y anexos.*) *Diego M. Argüello and Antonio Melita.* Arch. de oftal. de Buenos Aires 24: 116–22, June-July 1949.

Owing to the special sensitivity of nerve tissue to the rabies virus, the retina and optic nerve of experimental animals show a marked reaction to subdural injections of the virus. The choroid also has an abundance of nerve fibers and reacts positively in 40 to 58% of experiments. The vitreous is less sensitive than the choroid and the crystalline lens. Containing no vessels or nerves, it was least affected. Some writers believe that the aqueous humor is affected, but this has been denied by Relinger. The virus is also occasionally found in the nerve ends in the lachrymal gland and occasionally also in Harder's gland.

A child who had been bitten on the right side of the face, developed facial paralysis on this side, with lagophthalmus and concomitant keratitis. There was a discrete mydriasis and no abnormalities in the eye grounds. An adult who had received Fermis vaccine nevertheless contracted the disease, with hydrophobia, aerophobia, intense photophobia, slight conjunctival congestion and localized retinal hemorrhages in various sectors.

A review of the literature indicates that mydriasis, paralysis of the internal rectus, exorbitism and nystagmus are sometimes followed by ocular disturbances and coma, with hyperemia of the disc, venous dilatation, and acute chorioretinitis and retinal hyperemia. In some cases the primary lesion appears in the eyelids or conjunctiva.

A routine study of the eyegrounds of rabbits with preliminary dilatation of the pupils with atropine and injection of the rabies virus re-

vealed choroid hemorrhages in some. Some attenuated neuritic forms of rabies have also been described. The seventh nerve is particularly sensitive to the virus. Symptoms develop from 11 to 30 days after onset of treatment. They may be unilateral or bilateral and are often preceded by headache of neuralgic type. Motor disturbances may include lagophthalmos, ptosis, intrinsic and extrinsic paralysis. A review of the literature reveals only a few reports on ocular complications of rabies treatment. The present writer observed also sensory disturbances such as photophobia, pain referred to the eye and periorbital region, photopsias, etc. One patient suffered from intraocular optic neuritis, a condition which has been mentioned also by other writers. The course of these disturbances is benign and they are of short duration. 11 references.

Ocular Symptomatology of the Sino-Carotid Syndrome. (*Symptomatologie oculaire du syndrome sino-carotidien*). *A Franceschetti and M. Doret, Geneva, Switzerland.* Rev. méd. Suisse rom. 69: 603–12, Sept. 25, 1949.

The authors describe 4 cases of sino-carotid hyperreflexia with ocular manifestations. The first, a woman of 47, presented a history of continual headache of several months' duration, a variable decrease of visual acuity, concentric narrowing of the visual fields in spiral form, augmentation of the angioscotomata and variable changes of the retinal arterial pressure. Atropine produced a temporary cure and prevented the aggravation of symptoms which usually followed carotid massage. The authors stress the necessity of this clinical test of Tschermak in all cases of suspected circulatory disequilibrium of sino-carotid origin. The second, a woman of 31, presented a similar clinical picture—the classical syndrome of sino-carotid hyperreflexia accompanied by headache and concentric spiral reduction of visual fields—also temporarily alleviated by atropine. On the basis of these two observations, the authors stress the chronicity of certain symptoms present between he periods of crisis. They also indicate an analogy between this picture and the chronic and acute symptoms of the post-traumatic meso-diencephalic syndrome, relating the symptoms to the particular vascularity of this area and its sensitivity to vasomotor disturbances. Apart from the acute visual clouding and temporary blindness in these "cerebral types" of sino-carotid hyperreflexia, one often notes a permanent, variable decrease of vision, a concentric narrowing of visual fields, an increase of angioscotomata and a hypotension of retinal arteries with excessive or discordant variations following a change of head or body position.

Other graver ocular manifestations may occur, such as thrombosis of the central retinal vein, which may be the first sign of the disorder

because of its concomitant sudden loss of vision. A third case, a man of 84 years, with a history of sino-carotid hyperreflexia, presented an important decrease of vision of several days' duration previous to entering the clinic. A classical picture of central venous thrombosis was present. One is tempted to ascribe this to the circulatory disequilibrium subsequent to the hyperreflexia. Later, the patient developed a glaucoma of the affected eye. A fourth case, originally described by Streiff, is reported as presenting a glaucoma and thrombosis of the central vein of the left eye after years of characteristic vascular instability.

In conclusion, the authors restate the ocular derangements which may follow disequilibria of the cerebral circulation, particularly in sino-carotid hyperreflexia—diverse functional and anatomical changes, *i.e.* temporary or permanent diminution of visual acuity, narrowing of visual fields, disequilibrium of retinal arterial pressure, glaucoma and thrombosis of the central retinal vein. Thus, collaboration between internist and ophthalmologist may assist in certain diagnoses and may lead to adequate therapy.—*Author's abstract.*

Contribution to Data on Sight Disturbances Caused by Proliferation of Pigment Epithelium. *Magda Radnót, Budapest, Hungary.* Brit. J. Ophthal. 32: 423–426, July 1948.

Pigment deposits appear on the lens capsule in iridocyclitis, glaucoma, and in chronic use of pilocarpine. Deposits have been described in the pupillary space following cataract extraction.

A case is described of an 83-year-old woman who had an intracapsular cataract extraction with peripheral iridectomy followed by iris hemorrhage which did not absorb in the ten days the patient was treated clinically. Ten weeks later a simple brown pigmentation was seen to cover the pupillary surface of the vitreous in a moss-like layer. It is postulated that the pigment epithelium had grown over the vitreous surface. 2 references.

Xeroderma Pigmentosa with Affection of the Eye. *Johan Sachs, Norway.* Brit. J. Ophthal. 32: 398–411, July 1948.

Three cases of xeroderma pigmentosa with ocular manifestations are recorded.

Case 1 was a 17-year-old girl who had "freckles" on her face and extremities which were painful when sunburned. A limbal tumor, right eye, was removed for cosmetic reasons. This recurred within two years. The tumor was pigmented and lay in the cornea. The skin showed pronounced lentigines, a form of pigmented naevi. The patient was treated with 4000 r X-ray therapy to the ocular tumor which subsequently disappeared. Numerous basal cell carcinomas and non-differ-

entiated carcinomas have appeared on various parts of her body and have been irradiated successfully. She is now 27 years of age.

Case 2 was a 32-year-old man, brother of Case 1. He too had widespread pigmented naevi which were sensitive to sunlight. He had developed a tumor of the neck at age 27 and numerous tumors since, all successfully irradiated. A small vascular growth was present at nine o'clock near the limbus, right eye. This was interpreted as a part of the general skin disease.

Case 3 was an 18-year-old white male who developed a tiny lesion at the limbus of the left eye at age 10. This was considered benign and the patient was not seen until age 18 when the entire left eye had become covered by tumor. He developed telangiectases, angiomas and naevi over the face and neck. A small tumor was developing on the right eye. The patient refused operation and died of carcinomatosis at age 21.

The disease is hereditary and probably due to a recessive gene. Members of afflicted families should avoid consanguineous marriages. 10 figures. 1 chart. 3 references.

Denig's Operation for Trachomatous Pannus. *N. Pines, London.* Brit. J. Ophthal. 32: 385-394, July 1948.

Denig's operation for trachomatous pannus is suitable only for the third or fourth stages of trachoma by MacCallan's scheme of grading. The bulbar conjunctiva is cut from the libus toward the periphery, the wound being half as wide and as long as the base of the pannus. All tissue is removed, baring the episclera. A lip mucosa graft is cut slightly larger than the conjunctival graft and all fat is removed. This leaves only mucosa and submucosa. The graft is fixed with silk. The mode of action is open to much debate. Filatov reports success with cadaver conjunctiva. Skin is often used.

Four cases are presented. The first was between third and fourth stage trachoma with partial distichiasis. A graft of lip mucosa was made on the right lower lid for distichiasis and from ten to two o'clock in the right eye for pannus. Both operations were successful and mucosa is recognizable after nine years.

The second case suffered from recurrent trachomatous pannus in both eyes. A Denig's graft was performed in the right eye 12 years ago and was successful despite a central portion slough. A part of the graft was biopsied after 12 years and still showed lip mucosa characteristics. A similar operation was performed on the other eye 9 years ago. Both eyes have remained quiet.

The third case developed pannus crassus of the cornea associated with an epithelioma of the upper lid. After Denig's operation from eleven to two o'clock, vision rose in three weeks from counting fingers to 4/60. The pannus recurred and x-ray therapy aggravated the condition.

The fourth case presented trachoma chronicum cicatricum, distichiasis, a tremendous serpiginous ulcer of the right eye, and chronic dacryocystitis. After removal of the lachrymal sac and Denig's graft from ten to four o'clock across the cornea and ulcer, the eye healed but vision was lost from a leukoma. Microscopic examination of the graft 19 years later revealed lip mucosa.

More frequent use of mucosa grafts is advocated in plastic operations in trachomatous eyes. 8 references. 2 figures.

Traumatic Surgery of the Eye. *Harold G. Scheie, Philadelphia, Pa.* Presented at the American Academy of Ophthalmology and Otolaryngology, 1949.

The surgical treatment of injuries to the eye and adnexa can best be divided into immediate and definitive. Immediate treatment is mainly prophylactic and aimed at preventing further injury than that already caused by the initial trauma and to prevent, as far as possible, future complications. The injured eye should immediately be immobilized, a condition best obtained by patching both eyes and prescribing bed rest. Attempt should not be made to remove blood clots or foreign bodies without adequate anesthesia, good akinesia and proper lighting, for fear of converting a simple wound into one with extensive herniation of ocular contents. Atropine is advisable in most cases. Chemotherapy or antibiotic therapy should be instituted immediately, preferably both streptomycin and penicillin given intramuscularly. Tetanus antitoxin or a stimulating dose of tetanus toxoid should be given after any penetrating wound of the eye.

The second phase of the treatment of ocular injuries, as a rule, falls strictly within the domain of the ophthalmic surgeon. All of the precautionary measures just mentioned must be continued. The eye should be carefully evaluated to avoid hurried and ill considered treatment. As a rule, time is not a great factor for there is no danger of shock or exsanguinating hemorrhage. Infection usually is prevented by antibiotics or chemotherapy. This permits careful evaluation of the extent of the injury and leads to better judgment and skill in management.

The need for correct management is nowhere better exemplified than in injuries to the eyelid. Severe deformity requiring months of plastic surgery can be prevented by proper initial care. The skin of the eyelids and conjunctiva can be mobilized and shifted readily because it has an excellent blood supply and resists infection while remaining viable. Tarsorrhaphy, canthoplasty and sliding grafts can be used to approximate torn borders of the lid to each other, which even in the more severe injuries protects the eyeball and serves as a basis for future reconstructive surgery. If the torn edges of an eyelid are not joined, the remnants of the orbicularis muscle undergo contracture. What might have been

originally half an eyelid or even more, eventually becomes only a small tag of scar tissue laterally or medially, leaving a very extensive notch defect. If, on the other hand, the lid remnant had been utilized properly, all deformity might have been prevented.

The lid margin should be restored in all instances. If only a simple laceration, the wound edges can be sutured directly, or, if the tissue loss is extensive, by utilizing sliding flaps from the canthus. If there is any question of tension or separation, a tarsorrhaphy should be accomplished, fusing the opposing lid surfaces.

Injuries to the canalicular portion of the lower lid are problems because of the difficulty of repair of the canaliculus and of the medial canthal ligament. The torn ends of the canaliculus can be joined over a silver probe or by utilizing some other similar technic. Because the medial canthal ligament is difficult to restore, it is often helpful to substitute a strip of tarsal tissue taken from the lower tarsal plate. A strip about 2 mm. wide is dissected from the lower border of the tarsal plate retaining its attachment to the plate medially. The free end is then sutured to the anterior lacrimal crest to provide a firm anchorage for the torn end of the eyelid. If this is not done, the pull of the orbicularis muscle separates the repaired canaliculus which withstands no traction.

Management of injuries to the eyeball requires great judgment. The possibility of an intraocular foreign body must always be considered. Careful examination, including roentgen studies and the Burman localizer, are in order before any such wound is treated.

Many factors influence the surgical management of injuries to the globe, among which are the type and size of the wound, location of the wound, the presence or absence of a foreign body, the presence of lens injury, intraocular hemorrhage and others. Simple corneal wounds, with no prolapse of tissue, if small, can be left untreated. A smooth pressure dressing might lead to smoother healing. Atropine or pilocarpine may be employed to retract the iris from the wound depending upon its location. Larger wounds should be closed either by direct corneal sutures, a conjunctival flap or both. The safer method is probably the conjunctival flap, lens injury being less likely. Scleral wounds are best closed by fine atraumatic interrupted silk sutures. No objection could be raised to the use of catgut except that the needles are, as a rule, coarser and more difficult to place, predisposing to vitreous loss.

Lacerations of the cornea or sclera which are accompanied by intraocular damage are much more difficult to manage. Iris prolapse, as a rule, should be excised and the corneal wound closed as early as is feasible. However, if the prolapse is associated with severe lens injury, repair of the iris prolapse should be delayed even for as long as two or three days. This allows digestion of the lens, permitting linear extraction at the time of excision of the iris and repair of the wound. No

difficulty will be encountered in freeing the iris from the lips of the wound after this length of time. If, on the other hand, the iris is immediately excised and the wound covered by a conjunctival flap or suture and the lens allowed to remain, the cataractous material becomes highly irritating and predisposes to endophthalmitis phacoanaphylactica, secondary infection and secondary glaucoma. It is difficult to bring oneself to attribute these complications and delayed recovery to the irritation of lens material when the picture is already so obscured by the trauma of the original injury plus a subsequent operative procedure. One is, therefore, loath to reenter such an eye to remove the lens material. Such neglect very frequently results in calamity for the eye.

The prognosis for wounds over the ciliary body must always be guarded. The outlook is best in clean wounds of the ciliary body where there has been little or no vitreous hemorrhage. A wound perpendicular to the limbus results in less bleeding than a jagged wound or one parallel with the limbus because of the nature of the blood supply of the ciliary body where the vessels run from before backward parallel to each other. Large jagged wounds with prolapse of the ciliary body usually require enucleation. Enucleation, however, need not be hurried because infection is easily controlled by chemotherapy or antibiotics, and there is no danger of shock or hemorrhage. Operation should be deferred until all possibility of associated intracranial injury is excluded. This frequently necessitates having careful x-ray studies of the skull.

In more questionable cases, the indications for enucleation are not clear and the problem of sympathetic ophthalmia is of great gravity. The ophthalmologist gains some reassurance from knowing that this rarely, if ever, occurs less than two weeks after injury. It is also emphasized that eighty percent of cases occur within three months after the injury, the most dangerous time, therefore, being from the fourth to the eighth week. Any eye, therefore, can be observed safely for two weeks following an injury. After this time, until the third month, extreme care is urged. The most dangerous type of eye is one in which wound healing has been interfered with by incarceration of the iris, ciliary body, or lens material, or by retention of a foreign body within the eye. If such an eye develops subacute inflammation and begins to soften, it presents the most common exciting cause of sympathetic ophthalmia and should be removed. The only treatment for sympathetic ophthalmia is prevention.

The management of intraocular foreign bodies is an extremely complex subject. Emphasis is placed on always suspecting their presence. When demonstrated, localization can be carried out by whatever means one prefers. Magnet extraction is accomplished by either the anterior or posterior route. The posterior route should be elected if the foreign

body is posterior to the lens, and the anterior route, if it is in the anterior chamber. When utilizing the posterior route the incision is carried only to the uveal tract, the foreign body making its own opening through the choroid or pars plana as it is attracted to the magnet. If posterior to the pars plana, diathermy punctures should be made to prevent subsequent retinal detachment. The most feared complications are intra-ocular hemorrhage, infection and retinal detachment.

Pathology of Eye Injuries. *Merrill J. Reeh, Portland, Ore.* Presented at the American Academy of Ophthalmology and Otolaryngology, 1949.

Injuries to the globe may be divided into two groups, namely, non-perforating and perforating injuries. For the most part non-perforating injuries are minor in nature. The contusion, however, is one notable exception. It may be associated with numerous pathologic changes, many of which are of serious nature and constitute a threat to the sight of the involved globe.

Perforating wounds of the globe are all potentially serious and frequently require immediate surgical care. There are numerous complications which may follow immediately, during convalescence, or late. These must be fully understood in order for one to be cognizant of the nature and extent of injury which exists, and be able to administer proper care throughout the entire period of management. As a general rule globes which have suffered double perforations, severe degrees of prolapse of intra-ocular contents, severe intra-ocular hemorrhage, or intra-ocular infection have an unfavorable prognosis. A few perforating injuries will result in fulminating panophthalmitis with nearly complete destruction of the ocular contents.

Improper wound closure may result in a soft, fistulous eye with subsequent ingrowth of surface stratified epithelium. Closure of traumatic and surgical wounds will be discussed. Ingrowth of fibrous tissue, and endothelial changes must also be considered in the differential diagnosis.

Foreign bodies are seldom tolerated by the eye. Much depends upon the substance of the foreign body, the extent of the initial injury, and the complications which develop. Unfortunately many intra-ocular foreign bodies are missed each year because of the ophthalmologist's lack of awareness of this possibility in association with apparently minor eye injuries. The value of an accurate history with particular reference to the mechanism of injury was emphasized. In general organic substances are followed by acute inflammations. Copper and brass are tolerated poorly. Iron and steel fragments may in themselves result in little harm but subsequent oxidation and dissemination of iron produces extensive degeneration. It is true that some foreign

bodies are tolerated for many years with minimal reaction; however, they are potentially dangerous and must be observed.

Sympathetic ophthalmia is always a potential threat in cases of perforating wounds of the globe. It is one of the tragedies encountered in the practice of ophthalmology. The pathologic change in both the sympathogenic and sympathizing eye is that of a granulomatous inflammation. In general it tends to involve the entire uveal tract without destruction of neighboring tissues. Necrosis is seldom encountered.

Immediate surgical intervention, adequate postoperative care, proper handling of intraocular foreign bodies, and immediate enucleation of hopelessly injured eyes is essential, in the prevention of sympathetic ophthalmia.—*Author's abstract.*

Improving Technic by Means of Rabbit Surgery. *Frank W. Newell, Chicago, Ill.* Presented at the American Academy of Ophthalmology and Otolaryngology, 1949.

Rabbit surgery offers an important and convenient means for improving one's operative technic and for developing new surgical procedures. Living animals or the heads of rabbits recently killed for food or laboratory studies may be used. It is essential that living animals be anesthetized completely during the surgical procedure and destroyed immediately following surgery unless an aseptic technic has been adhered to throughout the operation.

Only local medication is used preoperatively. Eserine ointment is instilled prior to all procedures requiring a limbal incision to prevent prolapse of the iris. Three per cent atropine solution and 10% neosynephrine are used prior to a keratoplasty. All other procedures may be done without preliminary medication.

Dial with urethane (diallyl-barbituric acid, Ciba) is administered intravenously for general anesthesia. We no longer use a precalculated dose of the anesthetic but inject about 1.0 cc. over a two minute period and then wait approximately five minutes. Then 0.10 cc. is injected cautiously every 30 seconds until the corneal reflex disappears. The greatest danger in the use of Dial is a respiratory arrest, but this is compensated for by a prolonged action. The general anesthesia is always supplemented with 0.5 per cent tetracaine topically. Euthanasia is obtained with intravenous injection of a saturated solution of magnesium sulfate.

The hair and skin around the eye to be operated are not washed. The vibrissae are cut and a single drop of silver protein solution is instilled and the fornices are then irrigated with 1.5% saline solution. The eye is draped with a sterile handkerchief. Sterile gowns and gloves are not used.

The operating table is as simple as possible, with screw eyes used

for extension of the animal and with a permanently attached bar for holding the head holder. A Czermak type head holder (Harvard Apparatus Co., Dover, Mass.) or other means of fixation, is almost essential for good surgery. The eye is exposed with the usual type of lid speculum or with lid sutures.

All of the usual surgical procedures may be performed on rabbits with the exception of surgery of the lachrymal apparatus. Because of the great delicacy of rabbit tissues it is necessary to use sharp and proper instruments rather than operating room discards.

If the animals are to be followed postoperatively, they must be given the same conscientious attention and treatment as is given to human beings. Usually no bandages are applied nor are the lids sutured. Ocular sutures are removed at the usual time, using ether inhalation anesthesia.

Local regulations regarding animal surgery may be obtained from city or state health departments. The rules of the National Institute of Health regarding anesthesia, asepsis, choice of procedure and convalescence must be followed.—*Author's abstract.*

Malformations Within the Eye. *Ralph I. Lloyd, Brooklyn, N. Y.* Presented at the American Academy of Ophthalmology and Otolaryngology, 1949.

There are three critical phases in the development of the eye: formation and closure of the optic fissure; formation and regression of the hyaloid artery and the Bergmeister papilla; and development of the chamber angle, with the canal of Schlemm, and iris. In addition to the defects traceable to variations in these complicated processes there are other defects, like extrapapillary coloboma, albinism, choroideremia and melanosis, which may be dominant or recessive hereditary characteristics.

Coloboma of the Disk and Choroid Conus, Pockets and Hernias of the Nerve Sheath and Meningoceles, Extrapapillary Coloboma: Colobomas of the disk and choroid result from defective closure of the optic fissure and appear in all degrees from shallow dents in the nerve head up to large defects in the iris, choroid and optic nerve.

Conus and other defects of the group are often associated with defective closure of posterior portion of the optic fissure, but faulty insertion of the nerve stem in the vesicle and defective development of the pigment layer of the retina about the disk, which in turn seriously affect the development of the choroid and sclera, must also be considered. The minimal error is the conus or crescent adjoining the disk without change in level.

Coloboma is a more serious error with definite change in level due to defective scleral development which may amount to staphyloma. Conus is due to failure of the pigment layer of the retina to reach the disk

margin with defective or rudimentary choroid exposing a crescent of sclera. On the opposite side of the disk, the choroid, retina and sclera overhang the disk giving the appearance of a shortened diameter. This crescent is located below in 67%; internally in 8.1%; in and down in 7.3%; out and down in 5.4%; and up and out in 7.2% (Vossius).

In contrast, the acquired myopic crescent is temporal in 79%; annular in 11%; above in 4%; and below in 6% (Hertel). The lamina cribrosa arises from the choroid and, if the choroid is lacking at some point, there may be defective attachment of the lamina. This may affect the whole nerve head, producing coloboma of the nerve head; or if local, a pocket at one side of the nerve, which in extreme cases may communicate during fetal life with a dilatation of the dural covering of the nerve, meningocele. In these pockets imperfect bits of retina and choroid form pigment deposits. In extreme cases, the lamina may be entirely missing, with the choroid covering the nerve head, or the pigment layer of the retina may surround the nerve to form its sheath. The nerve fibers dip into these depressions in the nerve head and errors of development such as nevi in the macular area may result. In conus with scleral bulging, the difference in refraction of the macula and a corresponding point opposite may amount to 12 diopters. The choroid is thin, sclerosis of the choroidal vessels occurs early especially in the conus, and the picture is similar to that in malignant myopia of adults.

Bergmeister's Papilla, Hyaloid Artery and Tunica Vasculosa Lentis: The lens develops within a vascular capsule supplied originally by the hyaloid artery arising from a cone of embryonic tissue on the optic disk, Bergmeister's papilla. The lens moves forward as it develops, stretching the branches of the artery until they atrophy about the seventh month, at which time the ciliary circulation becomes the permanent supply. The artery and the Bergmeister papilla should disappear, but often some trace of them may be seen on the disk, especially with yellow light. The embryonic tissue remaining on the disk may determine whether details of the cribrum are plainer to see but physiologic cupping of the disk and cornus depends on closing of the optic fissure. Variations in this complicated process are diagnostically important.

Persistent hyaloid artery may be merely a slender filament from the disk to the posterior surface of the lens, a little below and internal to the posterior pole. More pronounced deviations affect the vision. At the anterior attached end, there may be a biconvex disk of tissue originally described as pseudophakia fibrosa of Czermak.

Functioning vessels may run from the mass of the ciliary vessels. Some of this type show retinal detachment which, if complete, shrouds the hyaloid artery. These eyes are underdeveloped with small lenses, long slender ciliary processes, and shallow anterior chambers. Of late years this type of pseudoglioma is recognized and is found more often in premature infants. Clinically the diagnosis depends upon the cold

grey color of the tissue applied to the posterior lens surface and not located in the vitreous, a functioning vessel may be seen running out to the ciliary region, and the iris retains some function. The eye is smaller than its fellow and the side of the face and the orbit may be underdeveloped. Buphthalmos may develop in such an eye. Seven of 24 eyes removed at Moorfields for glioma were of this type. If there is serious doubt of the diagnosis in a unilateral case it is wiser to remove the eye after consultation and explanation that such an eye is useless.

Vessel loops are relics of intrinsic circulation in Bergmeister's papilla. Four types are described: 1) Simple loop forward, both ends on the disk. 2) Complicated arrangements, both ends on disk. 3) Loop with one end connected with retinal vessels. 4) Loop with one end returning to disk and then continued as a retinal vessel.

A vessel loop seen with the ophthalmoscope and also examined microscopically by Wexler and Goldstein was only pervious in spots.

Prepapillary Cysts: These are very rare and vary from a mere button on the disk to a large mass of 8 to 10 disk diameters. The contents of the few seen were clear. With the binocular scope the contents and base of the cyst may be studied. In one case, a vascular loop within was seen changing its position with the eye movements. One would expect the cyst to arise from the center of the disk but those studied have been eccentrically based.

Persistent canal of Cloquet is seen in great variety. It may appear as two dilated extremities connected by a narrow bar. The anterior extremity is free with filaments, while most pronounced fundus changes are seen about the posterior attached end.

Ablatio falciformis congenita: A rarity, but tissue organized about a foreign body or after trauma with hemorrhage may require differentiation. The typical congenital defect is a semilunar fold of retina extending from the disk to the lens with the hyaloid artery in the free axial margin. The fundus may be much altered about the attached border, resembling effects of disease. The acquired forms do not reach the lens and a fine brushwork reaches out into the vitreous from the anterior extremity. Some of these resemble bolder forms of persistent canal of Cloquet. Familial occurrence is reported by Ida Mann and Weve.

Variations in blood supply: Most common is the cilioretinal vessel, which may function after occlusion of the main vessel stalk and gives the well-known slit field with an acuity of 20/20, which is of little use because of small field and loss of reading guide. Occasionally the central artery of the retina is replaced by a group of ciliary vessels entering the globe at the side of the optic nerve. The retinal vessels may be extremely tortuous and full in hyperopic eyes, simulating optic neuritis because of accompanying glial tissue.

Congenital vessel communications also have very full and tortuous

vessels without change in acuity. The acquired communications seen in angiomatosis retinae and after trauma and the dilatations associated with nevus flammeus of the face will require differentiation.

Pigment on the Optic Disk: Congenital pigmentation is often seen at the disk margin but heavy pigmentation is rare. Very rarely the whole disk may seem heavily pigmented, but microexamination of a few of these showed absence of the lamina cribrosa with the pigment layer of the retina forming a sheath for the nerve, while in others the choroid may continue across the disk. Ida Mann identifies two types of pigment: crystalline ectodermal from the neuroglia; and mesodermal and amorphous about the blood vessels like choroidal pigment. Clinically three types are seen: 1) dense sectors running over into the retina; 2) linear markings near disk margin; and 3) lacy pigment connected with the disk vessels.

Masses of opaque optic nerve fibers and remains of the Bergmeister papilla may be pigmented. Acquired pigmentation of the disk is easily separated because of the marked changes associated with the disease causing the deposit. Melanomas of the disk are easily diagnosed by their bulk but the question whether they are malignant or not may remain in doubt even after microscopic examination.

Opaque Optic Nerve Fiber: The myelin sheath of the optic nerve develops after birth, ending abruptly at the lamina cribrosa. If the sheath extends beyond this point, it is not a mere abnormality but is associated with other deviations that stamp the organ as physiologically inferior. In addition to alterations in the nerve fibers, myopia, conus and colobomas are often seen. If the amount is considerable, vision is poor even if the macular area is free. The opaque tissue is not nearly as dense as it seems, as has been shown by illuminating the eyeball from the pharynx with the Hartzell apparatus. Patches of this tissue have been seen to disappear in optic atrophy, glaucoma, tabes and embolus of the central artery. Stephenson examined 4212 school children and found the frequency to be .68% and three-fourths of these were bilateral.

Extrapapillary Colobona: The name given by Lindsay Johnson in 1890 to differentiate these defects from defects associated with closure of the optic cleft. He attributes them to defective development of the vascular layer in which the choroid develops. A few have since been examined under the microscope and the original defect placed in the pigment layer of the retina with secondary results in the choroid and sclera. Less pronounced changes in the inner retinal layers have been noted, which probably explains why these patients can do so much better than others with comparable defects in the macular area although unable to read print. They are often associated with congenital pigment groups resembling the footmarks of animals and other anomalies and are either unilateral or bilateral.

Retinal veins may pass over the lesions but Johnson says an artery never follows such a course. Ida Mann believes them due to intrauterine inflammation with three forms: 1) pigmented defects, 2) nonpigmented defects with ectasia of selera, and 3) defects associated with vessels of different layers of the embryo passing into the retina. Lesions are seen with the characteristics of all three groups. Toxoplasmosis affecting the embryonic eye is considered by some as the cause. Differential diagnosis must be made between extrapapillary coloboma and healed tuberculous lesions and the type of hereditary macular degeneration with thinning of the choroid in the macular area surrounded by pigment.

Angle of the anterior chamber contains not only important elements of the blood and nerve supply and trophic control but also the canal of Schlemm, essential to drainage of the eye. Congenital glaucoma is caused by absence of canal of Schlemm, blocking of the chamber angle by embryonic tissue or adherence of iris root to the corneal periphery. At about the twelfth week, the iris begins to appear from the ciliary region and gradually grows inward between the lens in its vascular capsule and the cornea. The pupil begins to form about the sixteenth week, but it is not unusual for some of the filaments of the vascular capsule to persist with one end attached to the circulus iridis minor and the other free, attached to the lens or continuous to the opposite side of the vascular ring. Occasionally strands adhere to the posterior corneal surface and require differentiation from leukoma adherens.

A very rare malformation is gerontoxon cornene posterius. A zone of embryonic tissue encircles the periphery of the cornea on its posterior surface with coarse strands passing over onto the anterior surface of the iris. The coarse elements ordinarily seen on the anterior surface of the iris are derived from the anterior vascular capsule and are especially prominent in this condition. Occasionally these same coarse elements of the vascular capsule may stand out from the iris, and the embryonic tissue of the pupil area may remain as a gauzy membrane. There may be a mass of embryonic tissue on the anterior lens surface with strands from the iris or a mass of pigment stars imbedded in a structureless membrane or as isolated bits.

Aniridia is partial or total and seriously affects the vision as other defects in the eye are present. Glaucoma, even in adult years, is the most common complication because the error implies a serious defect of the chamber angle and ciliary region and even of the retina. Ectropion uveae is not common but unless associated with other defects is not of importance. Korectopia is rare and varies from slits to deformed pupils. Iris cysts are extremely rare and may run in families. Patches of the posterior epithelial layer of the iris are raised by fluid and may not be seen when the pupil is contracted.

Congenital miosis is due to defective development of dilating fibers

with normal sphincter. This is seen as a part of arachnodactylia with bilateral dislocation of the lenses. In early life the rigidity is mild but, along with the degenerative changes in the anterior segment of the fundus, it becomes so pronounced that even atropine will not open the pupil.

Melanosis: This exceptionally rare condition varies from gobs of pigment in the sclera at the limbus to masses of iris pigment, complete change in the normal arrangement of iris pigment with marked pigmentation in the fundus or, perhaps, only small masses of pigment in the fundus. Alien collections of pigment are always suspect because they may suddenly increase in size and vascularity and become malignant. Of the groups mentioned above only the type with widespread pigment is considered dangerous. The isolated group in the fundus is innocent.

Albinism is a recessive hereditary characteristic with more or less failure of mesodermal pigment but only partial ectodermal deficiency. The whole body may be affected or the eye or the fundus only. If at all pronounced, nystagmus, astigmia, myopia, amblyopia and head nodding are associated. It was formerly held that dazzling was the cause of poor vision but Elschnig has shown defective differentiation of the macula. Dark adaptation is unaffected because of presence of the pigment in the outer retinal layer.

Heterochromia Iridis: True heterochromia is rarely seen, but acquired alteration of the iris color in one or both eyes is more common and is usually a manifestation of tuberculous uveitis, which runs a quiet course without ciliary redness but with vitreous opacities, recurrent crops of iris nodules, secondary cataract, and even glaucoma of the secondary type. Occasionally an acquired type is seen without evidence of tuberculous origin and with a much slower course. The congenital type is stationary.

Choroideremia: Originally described by Mauthner as a congenital defect, but it is now evident that only three of the cases reported and illustrated can be considered as such because the blood vessel arrangements are not duplicated by disease.

Most of the other cases are abiotrophies affecting males, transmitted by females with fundus lesions much like those of inherited syphilis but with good vision. A few cases of retinitis pigmentosa with the choroidal element of the fundus picture obscured by glial overgrowth have been described by Verhoeff. A fourth group with a normal macular area, retinal vessels and optic disk and exposed sclerosed choroidal vessels against a heavy pigmented background is sometimes included, but progressive choroidal atrophy is the preferred title.—*Author's abstract.*

Congenital Cyclopia and Orbital Cyst Together with other Developmental Anomalies on the Same Side of the Face. *F. Papolozy, Budapest, Hungary.* Brit. J. Ophthal. 32: 439–443, July 1948.

A case is described of congenital cyclopia with orbital cyst associated with hare lip, cleft palate and a proboscis-like structure all involving the right face of a newborn male child. The left face was normal.

The cyst and proboscis-like structure were excised and the child died of pneumonia. Histological studies of the soft parts of the orbit revealed retina, choroid, ciliary body, iris and lens structures actually making this a case of congenital anophthalmos. It is emphasized that the rudiments of the eyeball can nearly always be found making it impossible to distinguish extreme degrees of microphthalmos from anophthalmos. 4 references. 1 figure.

Chemotherapy. *Kenneth L. Roper, Chicago, Ill.* Presented at the American Academy of Ophthalmology and Otolaryngology, 1949.

The use of sulfonamides and the antibiotics have swept the board almost clean of the old therapeutic agents. They have made obsolete many agents, many of our methods of treatment, and much of our past thinking.

We are required less and less to treat by the trial and error method. In the past symptoms, rather than causes, were treated. Routine bacteriologic study of ocular infections has a very definite place in the practice of ophthalmology today, particularly in view of recent advances in chemotherapy.

Of our known lid infections, the staphylococcic infections are the most important and most troublesome. Certain infections are obviously staphylococcic, the common sty being undoubtedly 100% staphylococcic in origin. But whenever possible scrapings and cultures should be taken routinely because we have reasonably satisfactory specific therapy today.

Chemotherapy has no permanent value in lacrimal sac infections, but the important thing to remember is that abscess formation and perforation can be avoided in these cases by prompt use of chemotherapy.

Cellulitis of the orbit is generally associated with infection in the neighboring nasal sinuses, and with prompt chemotherapy we seldom get the chance to make a bacteriologic examination, since incision and drainage are seldom necessary.

Cavernous sinus thrombosis was nearly always a fatal disease, but not so with heroic doses of penicillin and sulfadiazine, since the organism is invariably Staphylococcus aureus.

In purulent conjunctivitis, such as gonococcal conjunctivitis, the systemic administration of penicillin seems to be the treatment of

choice. The sulfonamides are still highly effective in this disease, and the rapidity of the cures is astounding.

The typical example of our acute catarrhal or mucopurulent conjunctivitis is the so-called "pink-eye" produced by the Koch-Weeks bacillus. A virtually identical picture can be produced by the pneumococcus. Sulfonamide therapy is the most effective in these cases.

The most common cause of subacute conjunctivitis is the influenza bacillus and response to sulfonamide therapy is very gratifying.

Chronic catarrhal conjunctivitis is probably one of the most common eye infections seen in practice. Most patients simply tolerate their condition. Bacteriologic studies in these instances cannot be taken too seriously.

We have two conditions affecting the conjunctiva, characterized chiefly by lymphoid follicles—follicular conjunctivitis and trachoma. The finding of the cytoplasmic inclusion bodies is diagnostic of acute follicular conjunctivitis. Until the introduction of the sulfonamides, no treatment was of any avail.

Trachoma is a specific virus disease and the clinical diagnosis made from the follicle formations, pannus and cicatrization is quite satisfactory. Laboratory diagnosis is useful in atypical cases. The sulfonamides are capable of effecting a cure in pure trachoma without secondary infection. It is probably well to use copper sulfate locally.

Corneal ulcers can be divided into two main groups—marginal and central. The serpiginous ulcer due to the pneumococcus is an example of the latter. Curettage of all necrotic material, with sulfadiazine powder in the conjunctival sac as well as orally, is used. Penicillin is also very effective in pneumococcic ulcer. Its use topically is more important than when using the sulfonamides.

The treatment of most marginal ulcers consists in treatment of the associated conjunctivitis. One type of marginal ulcer is the ring ulcer. It is generally of endogenous nature and treatment consists of attention to the systemic disease concerned. In the exogenous cases the use of penicillin and sulfonamides locally seems to be sufficient, and cauterization is rarely indicated.

The chronic serpiginous, or Mooren's, ulcer still is an unsolved problem. Delimiting keratotomy, combined with repeated paracentesis, seems to be the treatment of choice.

Treatment of the interstitial keratitis of congenital lues should improve when further studies with penicillin therapy have been carried out.

Infections of the interior of the eye are chiefly endogenous in origin. The exogenous infections occur after surgical procedures and perforating injuries. Treatment of nonspecific uveitis remains unchanged—fever therapy being of greatest value. In dealing with uveitis, specific therapy is indicated in a few known etiologic types.

Most cases of endophthalmitis and panophthalmitis are due to either pneumococci or staphylococci. In early cases, chemotherapy offers some hope. There should be no hesitation about injecting penicillin directly into the globe. This should be combined with systemic treatment with both penicillin and the sulfonamides.

The treatment of virus infections has changed very little. In the common dendritic keratitis the virus remains localized in the corneal epithelium and is destroyed by curettage and cauterization.

Early transfusion of convalescent serum helps in cases of herpes zoster. Other treatments consist of x-ray over the ganglion, sodium iodide intravenously, and solution of posterior pituitary. Repeated vaccination with vaccinia virus has been reported to give good results.

Therapy in the treatment of epidemic keratoconjunctivitis seems still to be limited to the control of the secondary bacterial infection.—*Author's abstract.*

Book Reviews

How to Become a Doctor. *George R. Moon, A.B., M.A.* Published by the Blakiston Co., Philadelphia and Toronto. 131 Pages. Price $2.00.

The author suggests that preparatory school students who seriously contemplate studying medicine strive for good marks. While indicating a preference for subjects in the arts course, he advises the study of inorganic and organic chemistry, embryology, and to dissect some small animals during the college course.

Then follows a summary of the admission requirements of the leading medical schools of the United States and Canada, and explanation of how admission committees function, and a discussion of the medical curriculum. The costs of a medical education are indicated, such matters as housing, outside employment, and finally, internships and residencies are explained.

A short chapter is added which discusses the study of dentistry, pharmacy and veterinary medicine. The book offers a useful reference source for the student who must have many questions to ask regarding the study of medicine.

Textbook of Ophthalmology, Vol. IV. 1081 illustrations, including 71 in color. 4627 pages. *Sir W. Stewart Duke-Elder.* C. V. Mosby Co., 1949.

Duke-Elder's textbook of Ophthalmology, Volume IV, is another invaluable contribution by a remarkable ophthalmologist. Volume IV covers the neurology of vision as well as motor and optical anomalies. As with all of the writings of this genius, the style is flowing and yet the presentation of his ideas is concise and to the point.

The section on the neurology of vision covers the visual pathways,

under the chiasmal pathways, the basal visual pathways, and the intracerebral visual pathways.

Under Disorders of the Higher Visual Centres, the author takes up the organization of the brain, the visuo-sensory disorders and associational disorders and paroxysmal disorders. Under paroxysmal disorders, he includes migraine and epilepsy. This continues with Disorders of Perception and Psychogenic Disorders.

In Chapter 44, anomalies of the pupillary pathways are considered in great detail, beginning with the physiological anatomy of the pupillary pathways, muscular examination, and continuing the work with static pupillary anomalies, periodic phenomena and anomalies of the pupillary reflexes.

The illustrations which are so important in a work on neurology are beautifully done and the titles are unusually descriptive. The bibliography of the section on neurology is quite extensive and sufficiently recent.

Section XV which deals with Motor Anomalies of the Eyes, covers the subjects of anomalies of the ocular movements, anomalies of binocular fixation, concomitant squint, noncomitant squint, ocular deviations and nystagmus. This section makes a valuable addition to the complicated subject of motor anomalies and the inclusion of references to much of the recent literature adds value to the presentation.

The author's classification of acquired ophthalmoplegias of nervous origin is unusually well done and brings the subject to date. One of his best chapters is on the ocular deviations and is one which has received little attention in some of the recent publications. Chapter 50 on Pathological Nystagmus which concludes the work on motor anomalies is quite complete and authoritative although it is possible that some of the more recent references would have added to the value of the presentation.

The volume is concluded with section XVI on optical anomalies of the eye which covers errors of refraction, anomalies of accommodation, aniseikonia, eye-strain and visual hygiene and clinical optical appliances. The entire chapter is clearly presented, beautifully illustrated, authoritatively discussed and presented and brought quite up to date by the inclusion of more recent developments; *e.g.*, data concerning aniseikonia. The chapter on anomalies of accommodation is prefaced by a photograph of Alexander Duane who did so much to clarify this important subject. The more important phases of accommodation which have received attention in recent years are clearly presented and cover such a controversial subject as fatigue of accommodation. The Chapter on Eye Strain in Visual Hygiene is exceedingly important for all those interested in industry and the important subject of eye fatigue.

The final chapter of the book deals with Clinical Optical Appliances.

This is an important section not only from the standpoint of the historical value of the contribution but also because of its practical value in clinical examinations. Those interested in neuro-ophthalmology, motor anomalies or optical anomalies must have this important contribution to the literature of ophthalmology.

The Practice of Refraction. *Sir Stewart Duke-Elder.* St. Louis, C. V. Mosby Company, 1949. 309 pages. 216 figures. Index. $6.25.

The first edition was published in 1928, of which there have been five editions with the fourth edition being reprinted twice. All editions of Sir Duke-Elder's book on refraction have been by far the most popular books on this subject in recent years.

The fifth edition contains several alterations, since the fourth edition was little altered due to the exigencies of the war. The plan of the book has remained unchanged but new material has been added on several phases of refraction. The etiology and significance of myopia is regarded from a different point of view. Transient changes in refraction are added in reference to more recent drug therapy. The subject of aniseikonia is objectively evaluated and the newer instruments for its testing are illustrated. The description of the mechanism of accommodation has been revised according to the more recent concepts. The chapter on convergence has been amplified by classifying and describing the types of anomalies of convergence and introducing the concept of fatigue of convergence. Orthoptic treatment is tersely evaluated as to indications, methods, and results to be expected in the light of more recent experience. Description of streak retinoscopy and velonoskiascopy have been added and the section on refractometry enlarged.

The chapter on spectacles has been improved by presenting the theory of best formed lenses, effectivity of lenses and by making the section more complete by the addition of several recent developments.

Those familiar with the previous edition will recognize the figures of former editions and will find several clearly presented new instruments. New readers will benefit by the author's unusually excellent arrangement of the material and clear but concise presentation of the fundamentally important work in ophthalmology. In this edition, the subject is brought up to date by addition of the most recent advances in the field of refraction and allied subjects.

For the student who requires a clear and concise work for studying refraction and for the practitioner who wishes to become authoritatively informed on recent developments in this field, this book assumes first place.

The Role of Plastics in Ocular Prosthetic Devices. *F. W. Jardon, Southbridge, Mass.* Presented at the American Academy of Ophthalmology and Otolaryngology, 1949.

In the past few years plastics have assumed a leading role in ocular prosthetic devices. Not only have plastics practically replaced artificial eyes but in the new field of integrated implants the greater majority of these are of plastic or plastic in combination with metal devices for the adherence of muscles and tissue. To date methyl methacrylate has proven the most satisfactory of the plastic materials. When used as an artificial eye, providing the fit is correct and the plastic is properly polymerized, the eye is perfectly comfortable and there is no irritation. The same material can just as successfully be used as a completely buried implant or as an integrated implant. If implants made of methyl methacrylate are extruded the fault does not lie with the plastic but with its curing, the design, the size or possibly because of the clinical background of the patient.

The ability to mold plastic into any shape makes it possible to correct many socket and lid conditions by the prosthesis alone. Sunken upper lids, drooping upper and lower lids can often be corrected by using certain shapes and special peripheral outlines. Shifted implant spheres, sockets having skin grafts and other cases of plastic surgery may be fitted successfully by using a special impression technique.

Sockets which are so small that the lids will not hold a prosthesis may be fitted with a small eye and through the use of pressure bandages and gradually increasing the size of the prosthesis the eye may be finally worn comfortably and look well. A socket having much fatty tissue can also be enlarged by increasing the size of the prosthesis which gradually through pressure will displace the fatty tissue. Plastic eyes are particularly advantageous for children needing them, since the eye will not break and can be enlarged as the child develops.

Implants so far have been mainly designed for primary operations. With the successes to date with this type new design and operative techniques are being developed for the much larger field of secondary operations. Modifications and adaptations of existing implants continue for this operation as well as completely new designs.

OTORHINOLARYNGOLOGY

Otogenic Abscess. *Gilbert Phillips, Sydney, Australia.* M. J. Australia 2:426–29, Sept. 17, 1949.

Otogenic cerebral abscess is comparatively rare but is usually suspected in patients having an acute mastoid or middle ear infection who develop symptoms of meningitis, cerebral compression or focal neural disturbance. A careful neurological examination with check for papilledema, aphasia, Hutchinson pupil and pyramidal defects is necessary

for differential diagnosis. An electroencephalogram will show a slow wave dysrhythmia of delta frequency traversing the abscess if one is present. The cerebrospinal fluid shows an increased protein content in 90% of abscesses, accompanied by a pleocytosis with 5 to 50 cells per cubic millimeter, including many neutrophils. A minimal amount of cerebrospinal fluid should be withdrawn for examination if the pressure is high.

Patients with symptoms of cerebral compression are usually referred directly to the neurosurgeon for examination. The otorrhea must not be disregarded however and chronic abscess requires exclusion. The differential diagnosis may be difficult in an aphasic or comatose patient but may be accomplished as with the first group described.

Treatment with penicillin and sulfadiazine should be commenced early and the focus of infection drained. A burr opening should be made over the location of the suspected abscess, the dura opened, and a brain needle passed into the hemisphere. If no abscess is found, the needle should be pushed on into the ventricle, a ventriculogram made and the presence or absence of abscess definitely determined. If an abscess is found, the pus is aspirated and 1 or 2 ml. of a penicillin solution containing 10,000 units per ml. injected into the cavity for every 10 ml. of pus aspirated. Thorotrast 1 or 2 ml. is then injected and the needle removed. Repeat radiograms of the abscess cavity are then made and aspiration of pus and instillation of penicillin repeated as indicated. The infecting organism should be determined by cultures from the first pus aspirated. A formal operation for removal of the abscess wall should be performed as soon as all signs of abscess activity have ceased. This treatment cures about 80% of cases. 3 figures.

Plastic Correction of Congenital Atresia of the External Auditory Meatus Combined with Fenestration to Improve Hearing. *Urpo Siirala, Turku, Finland.* Acta Oto-laryng. 37: 307–14, Aug. 1949.

The number of surgically treated cases of congenital atresia of the external auditory meatus is not very large. This is due to the fact that —with a few exceptions—attempts at surgical correction of this developmental defect have been unsuccessful. The most usual operation method in congenital meatal atresia is to open the mastoid cells and the antrum and then attempt to produce, by plastic surgery and skin grafting, a patent canal to connect the antrum with the open air. By this measure, which gives the sound waves free access to the middle ear, it was hoped to improve the hearing.

On November 22, 1948, a 9-year-old girl needed correction of a congenitally malformed left ear and improvement of hearing. The girl's right ear was entirely normal. The left ear showed typical primary microtia. There was no auditory canal. Behind the tragus, only a

slight depression was seen in the place of the opening of the canal. The tragus was smaller than normal. X-ray examination showed the right ear to be normal with fairly extensive pneumatization, but in the left ear no bony meatus was seen and pneumatization was less extensive than on the right side. Hearing was normal in the right ear; the left ear heard the loud conversational voice *ad auriculam*. Audiometric testing showed that in the right ear both air and bone conduction were within normal limits. The chart for the left ear resembled that seen in otosclerosis: it was principally a conduction defect but bone conduction was also impaired, especially in the high tone frequencies. Air conduction averaged 66 db. in the speech frequencies, bone conduction 23 db. The eustachian tubes were patent. The patient thus had normal hearing in one ear. According to the generally accepted principles, an operation was not justified only to improve the hearing in this case. Yet there is also the question of appearance and the psychologic aspect, which has been stressed by Pattee, etc.

The operation was performed on December 12, 1948. Under local aethocaine anesthesia an incision was made behind the auricle, forming a rectangular skin flap later to become the back wall of the canal. In the area of the depression behind the tragus there was a cup-like cartilage plate under the skin. The mastoid cells were opened and an endaural incision was made behind the tragus where the opening of the canal was to be; through this incision dissection was carried forward through the subcutaneous tissue into the operation cavity. The cup-like cartilage plate immediately below the skin at the opening of the canal was freed, flattened and turned backward to support the back wall of the canal. The opening into the middle ear was widened. The incus was seen and when an attempt was made to remove it by disconnecting the incudomalleal articulation the latter was found to be ossified. When moving the incus it came away with the head of the malleus. The malleus broke at the neck. The handle was found to be attached to the wall of the tympanum.

After opening the antrum and removing the malleus and incus the patient's hearing was tested keeping a Barany box in the normal ear. There was no noticeable improvement: loud speech in the ear was heard as before the operation. The conclusion was formed that the fenestrae did not permit the sound waves to pass. Consequently, fenestration of the horizontal semicircular canal was resorted to under ether anesthesia. The fenestra and the walls of the operation cavity were covered with Thiersch grafts which were held in position by pieces of sponge immersed in penicillin solution. The rectangular skin flap behind the auricle was turned under it and sutured to the posterior edge of the endaural incision to form the back wall of the canal.

In this way a large canal had been constructed leading to the middle ear; it was lined with epithelium. The canal was curved, the convexity

being to the dorsal side. Therefore it was not possible to see the middle ear through the meatus, but it could be seen from behind and below the auricle. At the insertion of the auricle, between the skin graft turned under it and the surface of the mastoid process there remained a chink which could be seen only by pulling the auricle outward. Through this opening the operation cavity and the middle ear could be examined with an otoscope. This opening does not disturb the patient and is useful for cleaning the middle ear.

The result obtained in my opinion shows that

1) The plastic operation here used—of which I have found no description in the literature—makes possible the construction of a patent canal from behind the tragus to the middle ear. This results in considerable cosmetic improvement.

2) In cases of congenital meatal atresia, in which the opening of the middle ear and the removing of incus does not result in improved hearing, fenestration may be considered on the same basis as in otosclerosis.

3) A Thiersch graft can be used for covering the surgically produced fenestra. 2 figures.—*Author's abstract.*

Hazards of Intense Sound and Ultrasound. *Hallowell Davis, St. Louis, Mo. Horace O. Parrack and Donald H. Eldredge, Dayton, Ohio.* Ann. Otol. Rhin. & Laryng. 58: 732–38, Sept. 1949.

Present evidence does not indicate that air-borne ultrasonic vibrations constitute a practical hazard to hearing or produce any specific effects on the nervous system or sense organs. In general it is high intensities that are potentially hazardous, not high frequencies as such. Sounds above 120 db stimulate the sense of touch and may cause temporary and possibly permanent hearing loss. Levels above 140 db are painful to the ear and exposures to such levels without special protection should be avoided.

A frequency of 6500 c.p.s., at 159 db caused nearly intolerable pain to one subject and ruptured his ear drum during a five-minute exposure. Some high-tone hearing loss above 10,000 c.p.s. persisted after 14 weeks.

The vague subjective feelings of fatigue, annoyance, irritation, etc., induced by very intense sound or vibration may represent nature's first warning of the presence of a stress that is not yet severe enough to cause more objective manifestations. 5 references. 1 figure.—*Author's abstract.*

Early Detection of Middle Ear Malignancy. *Howard P. House, Los Angeles, Calif.* Ann. Otol. Rhin. & Laryng. 58: 789–97, Sept. 1949.

A review of the literature reveals several interesting facts: 1) Carcinoma of the middle ear is relatively rare; 2) This disease is superim-

posed on a chronically infected middle ear in the majority of cases; 3) In general, treatment of middle ear malignancy is disappointing.

Successful treatment of this disease, regardless of its location, depends on an early diagnosis. This is especially true in the middle ear, for by the time the lesion becomes grossly visible, invasion of the underlying bone has often already occurred. The mortality in these cases is then high, irrespective of treatment.

It is very difficult to differentiate clinically between granulations in chronic otitis media and early malignancy. Certainly, repeated biopsy of granulations in a chronically discharging ear to continually rule out carcinoma, is not feasible. Likewise, in view of the rarity of carcinoma of the middle ear, prophylactic radical mastoid surgery cannot be performed in all cases of chronic otitis media.

A study of the cytology of vaginal and bronchial secretions by the Papanicolaou technic has been of definite value in determining the presence of cancer before it becomes grossly visible. It is my opinion this same procedure applied to the exudate of a chronically discharging ear may provide the otologist with the means of making an early diagnosis of carcinoma in these cases. With this in mind the secretions of a number of chronically discharging ears were studied by this method.

Two case histories are described in the original article, in which the Papanicolaou technic seemed to be of definite value in the early detection of cancer of the middle ear. By carefully examining the postoperative secretions this same technic may also aid in determining whether the entire lesion was successfully excised at the time of surgery. Although conclusions cannot be drawn on two such cases, I report them simply to stimulate interest in this diagnostic approach to the problem of middle ear malignancy. 14 references. 6 figures.—*Author's abstract.*

The Development of the Otic Capsule in the Region of Surgical Fenestration. *Barry J. Anson, Chicago, Ill. and Theodore H. Bast, Madison, Wis.* Ann. Otol. Rhin. & Laryng. 58: 739–50, Sept. 1949.

The otic capsule differs from all other skeletal elements of the human body in respect to function, speed of development, schema of ossification, histologic fabric.

Functionally, the otic capsule serves as an osseous box to house protectively the organs of hearing and equilibrium.

Its development is dramatically rapid. The capsule attains adult dimensions when the fetus reaches the middle of its intra-uterine existence; in contrast, typical long bone continues to grow through a period of twenty or more years. While retaining fetal dimensions throughout life, the capsule becomes imbedded in a thick investment of periosteal bone.

Constitutionally, the otic capsule is unique. It is made up of numerous (totally, fourteen) ossification centers. Bone spreads almost con-

currently from the core of each of these originally independent centers, not, as in a typical long bone, from a point of initial ossification in the middle of a diaphysis (shaft) toward terminal epiphyses (at the extremities). Fusion between centers is peripheral and complete. There remain no zones of secondary, or "epiphyseal," growth such as regularly occur in a long bone. Fusion, with complete obliteration of fusion-lines between centers early converts the otic capsule into an osseous unit, a single bone. In this respect it differs from the cranium, whose constituent elements (parietal, frontal, etc.) continue to be at least partially separable along their boundaries of original sutural contiguity.

Histologically, too, the otic capsule is exceptional. Its outer layer, thickened to outbulk the contained capsule, becomes pneumatized through invasion of petrous bone by mucous membrane. The middle layer, most complex of the three, retains throughout life a considerable fraction of its primordial cartilage in the form of chondral islands imbedded in the endochondral bone; once produced, as a tissue of combination nature, it is never remade into haversian bone. The inner layer is simplest; it forms a mere shell for the labyrinthic canals, cochlea and vestibule.

The developmental time-tables of the three layers differ. The three layers are present in the midterm fetus (183 mm.); yet they spread at different rates, and in this phase of growth, the cochlear and canalicular portions are not alike. This matter requires elucidation.

The inner, or endosteal, layer appears in the fetus of 18 weeks (150 mm.); it is complete at midterm (183). This layer undergoes no increase in thickness and none in size as a covering for the periotic labyrinth. It fuses with the middle layer in late fetal life or at term.

In the middle layer, the intrachondrial bone appears before the endochondral. In both divisions of the capsule, cochlear and canalicular, intrachondrial bone appears in the fetus of 16 weeks (120 mm.). It never wholly disappears. This kind of bone attains maximum development in the fetus of 21 weeks (183 mm.). In the cochlear region the intrachondrial islands retain primoridal forms up to the 28th week (246 mm. stage). Thereupon, slow formation of endochondral bone is initiated (deposited upon the intrachondrial islands). At term, the process is suddenly accelerated, growth continuing through the first few years of infancy with such alacrity that the cochlear region of the infantile ear closely resembles that of an adult bone. In the canalicular region there is no such lag in development. From its first appearance, deposition of endochondral bone is rapid up to the age of 30 weeks (270 mm.), at which stage the adult condition is attained. It is never replaced by haversian bone. This layer remains distinctive in the adult.

The outer, or periosteal, layer is complete in the cochlear region earlier than in the canalicular division of the otic capsule. As an extracapsular addition, it continues to expand until the time of puberty—to

produce the petrous part of temporal bone. Beginning in the canalicular region, the periosteal bone of the otic capsule is invaded from the developing tympanic antrum. In the intercanalicular area, pneumatic spaces spread beyond the territory of the periosteal bone into the next layer, namely, the endochondral. So early is this invasion (beginning in the 28th week, or 246 mm. stage) that the endochondral bone never attains petrous character.

In the surgically important region of the otic capsule, namely, the tympanic wall of the lateral semicircular canal, the process of ossification is, in the early stages, the duplicate of that which operates to form any typical portion of the capsule: the three regular layers of bone are produced, to replace the primordial cartilage; together they undergo no expansion after once having fused along their contiguous surfaces. However, at approximately the stage of midterm, the process of ossification in the region of surgical fenestration begins to follow a special course. The capsule here consists of the three typical layers in the fetus of 18 weeks (150 mm.). At 22 weeks (193 mm.), the recently formed periosteal bone is being removed. So rapid is this process that in a 30-week (170 mm.) fetus, not only has the outer layer been resorbed, but varying amounts of the middle layer are removed, with some of the cartilage islands.

Following this resorption, new bone is added on the tympanic surface, to restore a smooth contour. As a consequence, the muco-periosteal membrane covers this secondary bone, instead of the original periosteal layer.

And so it is that the tympanic wall of the lateral canal, after having been eroded deeply on the external aspect, is rebuilt to assume regular structure and thickness. 4 references.—*Author's abstract.*

Otitis Hydrocephalus. *Austin T. Smith, Philadelphia, Pa. and Alvin Mirmelstein (Capt., M.C., A. U. S.)* Ann. Otol. Rhinol. Laryng. 58: 275–79, March 1949.

Otitic hydrocephalus commonly occurs in young people, especially children and adolescents. It is unknown whether the primary disturbance is an increased secretion or decreased absorption. Symptoms are headache, photophobia, vomiting, nystagmus, stupor, blurred vision and papilledema. It must be especially differentiated from brain abscess but also from cerebral tumor, encephalitis and aseptic meningitis. Treatment varies with the case. Some clear promptly after the first diagnostic lumbar puncture while the procedure may need to be repeated once or twice daily with others.

A case is reported in a 27-year-old man who had an acutely painful right ear of four days' duration. He had had recurrent drainage from this ear for about seven years. Examination showed tenderness over

the mastoid, zygoma and front of the ear. The white blood count was 16,600 with 92% polymorphonuclear leukocytes. Culture of the aural discharge showed *Bacillus proteus*, hemolytic *Staphylococcus aureus* and *Streptococcus fecalis*. Roentgenograms showed pronounced sclerosis of both mastoids but no bone destruction. Skull plates were negative.

Treatment was penicillin 100,000 units every three hours, sulfadiazine 4 Gm. initially and 1 Gm. every four hours, and streptomycin 0.5 Gm. every four hours. Fever, headache and toxemia increased and, six days after admission, he had chills and a rectal temperature of 104 F. Lumbar puncture showed a clear spinal fluid but pressure of 375 mm. of water. Chemical and eye examinations were normal. A right mastoidectomy showed scattered necrotic areas extending into the zygomatic area. The lateral sinus was inflamed and thickened. It was incised and a partial thrombus removed. Spinal fluid continued clear but the pressure was over 600 mm. of water the second postoperative day, dropping to 220 after removal of 25 cc. of fluid. Culture of the fluid was negative. The patient gradually improved, became afebrile, and was discharged but tired easily, had some headache and a spinal fluid pressure of 450 mm. water. The mastoid wound was slightly tender and began to discharge purulent fluid about ten weeks later. Neurological examination was negative but eye examination showed bilateral papillary edema secondary to increased intracranial pressure. An electroencephalogram and skull roentgenograms were normal. A right subtemporal decomposition was done and the brain punctured in 5 directions but no pus found. The papilledema was reduced and spinal fluid pressure 300 mm. water a month later. A combined lumbar-ventricular air encephalogram showed nothing abnormal except a slight shift of the lateral ventricle to the right. Otitic hydrocephalus was diagnosed. The mastoid was reopened two months later and granulation tissue in the antral region curetted out. He was discharged a week later, asymptomatic and with cerebrospinal fluid pressure of 230 mm. water. 1 reference.

Lightning as a Cause of Hearing Loss. *G. Brooks West, Jr., Cambridge, Md.* Laryngoscope. 59: 1350–54, Dec. 1949.

A number of bizarre lightning injuries are reviewed with special reference to trauma of the ears. One case of uncomplicated, unilateral hearing loss following lightning trauma is reported. Initial examination revealed no visual injury to the external ear or drum and final examination nine months after injury revealed the disability to be temporary with almost complete recovery. It is believed that the pathological process involved was one of edema involving the cochlea or auditory nerve or possibly the hearing cortex. No similar case was found in the literature. 9 references. 1 figure.—*Author's abstract.*

Detection of Noise Susceptible Ears. *D. E. Wheeler, Iowa City, Iowa.* Laryngoscope. 59: 1328–38, Dec. 1949.

The hypothesis is stated that damage in ears exposed to noise lies along a continuum, ranging from a reversible temporary loss for short exposures at moderate levels to permanent irreversible loss at excessive levels, such as those produced by blast or concussion. Within this continuum, individual ears respond differentially, giving reason to the belief that some individuals are susceptible to hearing impairment as a result of noise exposure. One possible mechanism, correlating physiological alterations with functional changes in acuity, is described. It is suggested that susceptibility may be detected in advance by exposing individuals to test noise, the criteria of susceptibility being 1) the magnitude of initial threshold shift and 2) the time required for recovery to pre-exposure threshold.

It is postulated that predictive tests must be shown to be valid by actual demonstration in noise environment; a technique is described. Apparatus for standardizing presentation of predictive tests is discussed. 9 references. 1 figure. 1 table.—*Author's abstract.*

The Fenestration Operation for Otosclerosis. A Survey of Experience with 75 Cases Together with a Study of the Effect of the Contralateral Ear. *DeGraaf Woodman, New York, N. Y.* Laryngoscope. 59: 1299–1319, Dec. 1949.

The history of the development of the fenestration operation is reviewed, with a survey of the experiences during the past three and a half years with 75 cases in which the method and modifications used are described. The complications encountered and their sequelae are noted, together with an autopsy report with histological findings in the temporal bone.

One case is presented showing the effect of infection on the postoperative hearing with a table of the decibel average for the critical frequencies before and after removal of the infection.

The method of reporting the end results of fenestration operations is discussed and the four classified groups used in the survey are described.

Improvement in bone conduction in the operated ear is mentioned and compared with similar findings and results of other operators.

The contralateral effect of improvement in the unoperated ear has been recorded by Sourdille, Kopetzky, Holmgren, Venker, and others. Both Sourdille and Holmgren have reported this effect as only early and transient. Holmgren reported contralateral improvement for both air and bone conduction checked by tuning fork tests.

The author's experience resulted in many cases which presented this early subjective contralateral improvement, together with a small

group (15%) which showed a definite (13.5 db) improvement in bone conduction which lasted during the first twelve months.

The contralateral and homolateral pathways to the cerebral cortex have been demonstrated by the recent work of Walzl. The contralateral effect in the case of the vestibular component of the eighth nerve has been demonstrated by the work of Cawthorne, Fitzgerald and Hallpike.

An attempt is made to draw an analogy between the tonic contralateral effect as seen in cases of the vestibular component and apply a similar explanation to the effect on the cochlear nuclei and their contralateral pathways which follows the fenestration operation.

It is the author's impression that there is a definite early improvement of hearing in the unoperated ear. This effect seems to be a central tonic response to the stimulation caused by the fenestration operation. This effect is compensatory and transient in character. The few cases of bone conduction gain in the unoperated ear may be explained as being coincidental. If they are the result of some central tonic action, the reason for their being so few in number may be based on the phasic quality of post-operative testing similar to that noted in preponderance tests of caloric nystagmus resulting from stimulation of the vestibular nuclei.

The series is too small for definite conclusions, but the results strongly suggest a central tonic contralateral effect. The attempt to explain this by drawing the analogy between the vestibular and cochlear components of the eighth nerve may be elaborated upon when studied in a larger series with more frequent audiometric testing for both early and late post-operative stages. 30 references. 3 figures. 1 table—*Author's abstract.*

Neurinoma of the Eighth Pair. (*Neurinoma del octavo par.*) *Raul Barrios, Santiago.* Revista de otorinolar. 9: 91–98, Sept. 1949.

A woman of 47 years of age had suffered for 4 years from characteristic symptoms of neurinoma of the eighth nerve with tinnitus, vertigo, occipital headache, weakness of the limbs on the right side, diminished hearing, uncertain gait and visual disturbances. For the past 6 months she also developed diplopia and a tremor in her right hand. The sense of taste was diminished but she had no difficulty in swallowing. It was noted that she inclined her head to the right side. The results of the various vestibular, roentgenographic and neurologic tests are given. The only treatment for this type of tumor is surgical, and since the results of attempted extirpation are usually poor, a palliative decompression operation is recommended. The patient showed improvement following the operation in the present instance but removal of a recurring tumor will be impossible owing to adhesions. Special attention is drawn to symptoms caused by involvement of the adjoining

cranial nerves. The tumor in the present case was found to be a large neurofibroma of the acoustic nerve, extending through the foramen magnum and to the second cervical vertebra. 6 references.

The Morphogenesis of Cholesteatoma of the Middle Ear. (*Ueber die Morphogenese des Mittelohrcholesteatoms.*) *Gerhard Eigler, Giessen.* HNO Beih. Zschr. Hals- Nasen-, u. Ohrenh. 1: 436–43, Heft 10, Aug. 1949.

The author argues that the cause of cholesteatoma is to be sought in the peculiar growth potential of the epidermis of the auricular canal as determined by the embryonic development of the latter rather than to any changes in the middle ear. The cord- and tube-like growth tendency of this epidermis beneath the muco-endostium in cholesteatoma must be determined in its cells and is relegated to the embryonic development of the secondary auricular canal. Ontogenetically there are two pneumatization principles in the aural region, one entodermal and one ectodermal. The two are interrelated. The factors which lead to renewed cavity formation after completed development of the auricular epidermis, include acute and chronic inflammations in the region of the middle ear, constitutional arrests in the development of the muco-endostium, prolonged negative pressure conditions in the middle ear and artificial and chemical irritants such as tar in the auricular canal. These factors can lead to development of cholesteatoma, when besides the above mentioned inhibition of pneumatization, two further prerequisites are fulfilled: the stimuli must lead to marginal defects and the defects must lie within undifferentiated mucoendostium. The reason for the higher ratio of males affected remains obscure. The sex incidence in the author's experience was 3 males to 1 female. This new conception is presented in the hope of stimulating fresh investigations. 55 references. 5 figures.

Endaural Surgery of the Temporal Bone. *Rear Adm. C. A. Swanson, M.C., USN, Washington, D. C.* Laryngoscope. 59: 984–1001. Sept. 1949.

Temporal bone surgery at the U. S. Naval Hospital, Bethesda, Md., has been routinely performed through an endaural approach for the past 3 years; adequate exposure through this approach has been obtained in all cases. The accessibility of remote recesses in the middle ear and hypotympanum, the absence of postoperative infection or scar, and the excellent drainage afforded by the endaural technique give it many advantages over the postauricular approach. Skin grafting of the mastoidectomy cavity helps to prevent excessive granulations in the middle ear.

The diagnosis of otosclerosis must usually be made by inference rather than upon pathognomonic symptoms or signs and therefore testing of speech reception as well as pure tone reception is a vital preoperative procedure. Further, an evaluation of the psychosomatic background of the patient is of great importance in determining the suitability for surgery. 21 references.—*Author's abstract.*

Monostotic Fibrous Dysplasia. Report of Two Cases. *S. L. Cooke (Col., M.C., U. S. A.) and W. H. Powers, Oak Park, Ill.* Arch. Otolaryng. 50: 319–29, Sept. 1949.

Fibrous dysplasia is a comparatively recent title for fibro-osseous disease. It is a benign tumor affecting one or several bones. The etiology is unknown. The spongy bone of the marrow cavity is replaced by whitish or red-specked rubbery tissue which is essentially fibrous in nature but contains scattered trabeculae or poorly formed primitive bone. Cyst formation may follow focal degeneration or hemorrhage. Pressure of the expanding tumor may cause the cortex to be thinned or partly absent.

The monostotic type of disease usually first manifests itself by a swelling over the involved area of superficial bones but may only be discovered on routine roentgen examination. There may be local tenderness or pain but this is not characteristic, the tumor frequently not being discovered for years. The bones most commonly involved are the tibia, femur, humerus, ulna and maxilla, only 9 cases of the last having been reported. Premature sexual maturation, skeletal growth and pathologic skin pigmentation, usually over the growth, are the most frequent abnormalities. The osseous lesions are generally confined to one limb or there is greater involvement of the bones on one side of the body than the other in the polyostotic variety. The blood chemistry is within normal limits in both forms of the disease. Roentgenographic findings are frequently difficult to interpret in the monostotic form, appearance of the bone varying with composition of the medullary tissue and bony cortex. The medullary cavity will appear radiolucent if filled with fibrous, somewhat cystic, rather than osseous tissue and will present a ground glass appearance if filled with ossified tissue. A multilocular appearance may be presented if the fibro-osseous tissue erodes through the cortex and causes the bone to become furrowed.

Roentgenograms are usually diagnostic with multiple bony involvement but not in the monostotic form because the single lesion is indistinguishable roentgenographically from bone cysts, enchondromas, giant cell tumors, etc. A biopsy is then necessary to distinguish them. The greatest difficulty in differential diagnosis is between polyostotic fibrous dysplasia, hyperparathyroidism, endochromatosis and Hand-Christian-Schuller disease. Maxillary lesions similar to the two reported cases

are often diagnosed as ossifying fibroma. The latter has been stated by various investigators to be a variation of fibrous dysplasia and not a separate entity, the only difference being that the trabeculae are spherical in ossifying fibroma and elongated in fibrous dysplasia. The prognosis for life is good in both types of the disease.

Treatment depends upon severity of symptoms. The disease may be eradicated in long bones, such as the tibia or femur, by thorough curettment and filling the cavity with autogenous bone chips. Resection is usually necessary for incapacitating lesions of the ribs or facial bones. A massive autogenous graft has been successfully used in weight-bearing bones. If possible, surgery should be avoided except for biopsy.

The case histories of 2 patients with fibrous dysplasia of the maxilla are presented. 12 references. 5 figures.

Two Cases of Epithelioma of the Larynx in Patients with Pulmonary Tuberculosis. (*Deux cas d'épithelioma laryngé chez les tuberculeux pulmonaires.*) *R. Pago and J. Mesnage.* Rev. laryng. Bord. 70: 549–51, Nov.–Dec. 1949.

In the first case reported, the patient, a man forty-nine years of age had been under treatment for pulmonary tuberculosis since 1947, and was in good general condition. He came under the authors' observation because of a swelling in the right side of the neck. There was slight difficulty in swallowing fluids, but no dysphonia or dyspnea. Examination and biopsy showed an epithelioma of the pyriform sinus and aryepiglottic fold on the right side with involvement of a cervical gland. In the second case, the patient had chronic pulmonary tuberculosis and was in poor general condition; he had had symptoms of laryngeal involvement which had recently become more severe, dysphonia, dysphagia and increasingly severe dyspnea. Streptomycin had been given, without any improvement of the laryngeal lesions. Examination showed tuberculosis of the larynx with multiple superficial ulcerations, and in addition a swelling on the right side in the region of the epiglottis; this proved to be a basal cell epithelioma.

The authors' observation of similar cases leads them to conclude that epithelioma of the larynx is not extremely rare in cases of pulmonary tuberculosis. In cases complicated by laryngeal tuberculosis, as in the second case, the epithelioma may develop in the region of the tuberculous lesions.

Cochleo-Vestibular Lesions in Acute Carbon Monoxide Poisoning. (*Les lésions cochléo-vestibulaires dans l'intoxication aigue à monoxyde de carbone.*) *H. Koumrouyan, University of Berne, Switzerland.* Pract. oto-rhino-laryng. 11: 307–21, Nov.–Dec. 1949.

A study was made of the cochlear and vestibular symptoms of 4

cases of acute carbon monoxide poisoning. Deafness and vertigo were evident as soon as the patient regained consciousness after exposure to the carbon monoxide. The deafness was of the inner ear type and was accompanied by tinnitus. The intensity of the tinnitus was proportional to the degree of hearing loss; if the hearing improved, the tinnitus diminished or disappeared entirely. If the hearing improves this occurs during the first month after exposure to carbon monoxide. Tuning fork tests always showed a shortening of the time of perception for C^3. The audiograms varied but showed deafness of the inner ear type, with hearing loss for the high rather than the low tones; in one case the hearing for only certain tones C^4 to C^5 was affected. Vestibular involvement was indicated by vertigo. Tests of vestibular function showed lack of agreement of the results of caloric and rotatory tests. In some instances the vertigo was not as severe as the objective tests of vestibular function would indicate. In every case of carbon monoxide poisoning the function of the acoustic nerve should be tested, as later the audiograms and vestibular tests may be the only means of proving the effects of the poisoning objectively when the patient complains of symptoms. 17 references. 12 figures.

Immediate and Total Suture in Acute Mastoiditis. (*Apropos de la suture immédiate et total dans les mastoidites aigues.*) M. H. *Aloin, Lyons, France.* Ann. d'oto-laryng. 66: 481–83, Sept. 1949.

The method of immediate complete closure of the mastoidectomy wound has been employed by the author for more than fifteen years, therefore for many years before the sulfonamides and penicillin were available. In recent years he has filled the cavity with a mixture of penicillin and a sulfonamide. In order for this method to be successful a total removal of the mastoid cells is essential. The dead space is obliterated by exact approximation of the planes of the suture, and the application of a pressure bandage. the dressing is first changed on the seventh day, as the cutaneous-periosteal flap formed by the suture begins to adhere to the walls of the cavity by the fifth day. As a rule healing is rapid so that the patient can be discharged from the hospital after the eighth day. The scar usually becomes almost invisible within a year. Hearing is better than with any other method of mastoidectomy.

Extensions of the Tympanic Cavity. *Victor Lambert, Manchester, England.* J. Laryng. & Otol. 63: 711–33, Dec. 1949.

A study is presented of the tympanic cavity in various lower vertebrates, in Old World monkeys, in great apes and in man, showing the structural changes in the various species and indicating why these changes are necessary in the air-containing cleft of the tympanum and

its extensions and upon what dictates of function these depend. No other study of the functional importance of the bulla has been published since the study of Cuvier of more than one hundred years ago, in which he showed that night-flying birds have an enormous inflation of the tympanic region because of the need for a quick sense of hearing. The diurnal birds, on the other hand, depend upon vision as their guiding sense and therefore have little or no inflation. In the primitive mammals the internal carotid artery is comparatively small, much less in size than the external carotid. In the primates the bulla observed in the primitive mammals is sacrificed to the advancing development of the internal carotid artery. In the higher primates the continuous cavities of the bulla and of the recessus epitympanicus have been reduced first to slightly septate chambers and finally to pneumatized areas of neighboring bones. Only so long as the internal carotid remains relatively small does the bulla remain highly developed. In the New World monkeys the internal carotid artery is fairly large, the inflation of the petrous apex no longer exists as a clear space but is best observed as pneumatized bone through which the carotid artery seeks its way into a discrete tunnel. In the marmoset this artery shows a great advancement and the bulla inflation arising from the eustachian tube before opening into the atrium communicates in some specimens with the atrium through the mouth of the eustachian tube and in others through pneumatized bone into the upper part of the atrium. However, in the Old World monkeys the greatly enlarged internal carotid artery passes through the apex of the petrous bone with no evidence of bulbous development, being here represented by a pneumatized area in the petrous apex which communicates with the tympanic extremity of the eustachian tube. The pneumatized petrous apex is noted also in the apes and in man, and is considered the last evidence of the typical mammalian bulla.

Lambert believes that all the extensions from the tympanic cavity present in the lower mammals were developed for the purpose of obtaining a high degree of auditory sense organ acuity. In the primates the internal carotid artery gradually enlarges in response to an ever-increasing brain and when eventually the high development of the cortex of the higher primates is achieved, this artery has usurped the entire area in which the primitive mammalian bulla developed. Thus, reduction of resonance chambers connected with the tympanic cavity is a harmonious process with enlargement of cortical auditory areas. Lambert agrees with Cuvier that mere auditory acuity is perhaps of the utmost importance as a life-saving mechanism in the primitive nocturnal animals, so that a highly efficient sound-receiving cortical mechanism is not necessary. In the lower mammals the mechanism is developed for a quantitative appreciation of sound and the cortical analysis is not necessarily well developed. The higher primates, however,

with an increasing cortical area devoted to psychical appreciation of sound require less efficiency of the auditory sense organ. The dominating influence of the auditory cortex in analysis of impulses from the ear is demonstrated in humans with varied acuity of hearing. The person with an alert mind can utilize blurred sound pictures whereas those of low grade intelligence are unable to compensate for their basic deafness. The replacement of the auditory bulla by pneumatization seems definitely related to auditory function, since the morphological changes taking place, from the simple bulbus eustachii to the middle-ear cleft, definitely tend to increase auditory selectivity as contrasted with auditory acuity. The tympanic areas of the various specimens are described and illustrated. 28 figures.

Modern Trends in Hare-Lip and Cleft Palate Surgery With a Review of 500 Cases. *Michael C. Oldfield, General Infirmary, Leeds.* Brit. J. Surg. 37 : 178-94, Oct. 1949.

The most important agents effecting the nasopharyngeal occlusion for formation of consonants are the levator palati sling arching the middle third of the soft palate and the "horse-shoe" muscle fibers which raise the Passavant's ridge. Therefore, in repair of a cleft palate, restoration and repair of the sling action of the levator is necessary. It is even more important to provide a rapidly arching and muscular soft palate than a long, flaccid one, for length is less important than muscularity and mobility. One in 600 babies born has lip and palate cleft, so that in England it is estimated that there are approximately 60,000 persons born with this defect. Eleven per cent of the 500 cases reported by Oldfield had a family history of the deformity. An incidence of 30 to 40% was noted in Fogh-Andersen's report of 703 patients. Although a recessive gene may be responsible in most cases, malnutrition or disease occurring in the first three months of the mother's pregnancy may be an additional causative factor. Hare-lip with or without cleft palate is more common in boys, whereas isolated cleft palate occurs more commonly in girls. If normal parents have a child with a hare-lip or hare-lip with cleft palate, there is a chance of only 5% that the next child will have the deformity but if one parent has the deformity the chance of deformity in the children is about 2% ; if a child is born with the affliction, there is a 15% chance that subsequent children will be so deformed. The incidence is high in children of two parents with hare-lip or cleft palate and hare-lip and marriage, with children, is contra-indicated. About 40% of the author's patients were first-born and 60% resulted from subsequent deliveries. In the series of Veau and of Fogh-Andersen, with about 1000 cases in each series, one-quarter had hare-lip alone, one quarter had cleft palate alone and one-half hare-lip and cleft palate together.

Veau has provided the best classification of cleft palate: I. Cleft of the soft palate alone. II. Cleft involving part of the hard palate as well as the soft palate. III. Unilateral cleft of the alveolus as well as the hard and soft palate, usually associated with unilateral hare-lip. IV. Bilateral cleft of the alveolus as well as the hard and soft palate, usually associated with bilateral hare-lip. In Type II there often appears to be a defect in the soft tissues associated with a congenital shortening of the palate, with a wide gap in the front of the cleft and sometimes an absence of the palatal bones. In Types III and IV the soft tissue is present but there has been a failure of fusion in the midline. In a high percentage of Type II both the mandible and maxilla are imperfectly developed and access at operation may be limited by the mandibular regression. Among 7 cases of mandibular regression, 2 newborn babies had tongues that periodically fell back, behind and above the lateral elements of the soft palate, causing sudden attacks of asphyxia when the baby was turned on its back. This tongue-swelling usually ceases by the third month.

Repair of the palate is best accomplished at the age of 18 months, so that the palate and nasopharyngeal valve are already effective when the child learns to speak. Under that age, operation does not produce any better speech results and at that age the mortality is 10 as compared with 2% during the second year. After the operation the patient lies on his side in the bed, anchored if necessary by a sandbag at the back. Penicillin 500,000 units is given intramuscularly twice a day for a week. The patient is usually discharged in 10 to 14 days. The patient then attends the speech clinic. It is important to refer all patients postoperatively to a dental surgeon so that the incisors can be guided during eruption into correct alignment. They should also be under the supervision of ear, nose and throat specialists since a high percentage have an infection of the eustachian tube and middle-ear disease.

During the last 15 years, in 205 cases of primary and 95 secondary cleft palate deformity, the speech results were excellent in 87 cases, good in 79 cases and bad in 17 cases. In 94.7% of the primary and in 80% of the secondary cases, closure was complete, the soft palate well arched and the nasopharyngeal valve competent. The various procedures used are described with excellent illustrations. The best results in repair of a cleft palate are by use of Veau's procedure as elaborated by Wardill and Kilner. The posterior palatine arteries should never be divided: the communicating artery between the anterior and posterior palatines should be divided between ligatures. Lateral packs should be removed after operation. Pharyngoplasty should not be associated with a primary repair of the palate but occasionally in secondary cases it may be performed using Wardill's technic. 29 references. 6 tables. 31 illustrations.

Otogenous Thrombophlebitis of the Lateral Sinus and Jugular Bulb. *A. Brownlie Smith, Edinburgh, Scotland.* J. Lar. Oto. Lond. 64: 12–16, Jan. 1950.

The diagnosis of otogenous thrombophlebitis of the lateral sinus could be exceedingly difficult, but since the introduction of penicillin, while the diagnosis has become even more difficult, the serious consequences of a faulty diagnosis have been diminished, as many cases of thrombophlebitis are now cured without ever having been diagnosed.

The author has a series of thirty cases, twenty-two of which were treated before the introduction of the antibiotics and eight after these were available. Of the twenty-two early cases, fourteen were complications of acute otitis media and eight of chronic otitis media, and of these twenty-two cases there were seven deaths, one due to the anaesthetic, giving a mortality rate of 32%. Of the eight later cases, for which antibiotic treatment was available, all of them were complications of chronic otitis media, and six of them were under the age of twelve years. All of them recovered after operation on the lateral sinus.

Three examples are given of the difficulty in the diagnosis, and of the complete masking of symptoms by the administration of penicillin.

It is in the cases of thrombophlebitis which do not respond, or only partially respond to treatment by the antibiotics that a diagnosis of thrombophlebitis is made today. In this group a partially effective treatment often modified the classical clinical picture of the disease to such an extent that the surgeon may be tempted to delay treatment for days or even weeks. The persistence of active concealed infection, even if of a low grade, should compel operative exploration. 2 figures.—*Author's abstract.*

Audiometric Studies of Presbyacusis. *T. J. Leisti, Turku, Finland.* Acta Otolar. Stockh. 37: 555–562, Dec. 1949.

The author has in his work studied age variations in auditory acuity in Finland. A brief survey is given of the audiometric technique and earlier investigations followed by a statistical study based on 902 audiograms taken from 451 persons aged 16 to 92 with normal ears. The results are shown in 5 tables and 12 diagrams. From these it is evident that the auditory acuity decreases very slowly with advancing age, the loss affecting mainly the high register from c^5 upwards. In the tone range below this limit the hearing loss is at first rather insignificant and becomes more pronounced only after the 50th year of life. The deterioration begins as early as at the age of 20 to 30 and seems to be from c^5 upwards in males more marked than in females. The results agree rather well with those obtained earlier. The tests were carried out under conditions at present possible in Finland (no sound

proof room) and therefore the results may be considered suitable for practical purposes. 9 references. 5 figures. 5 tables.—*Author's abstract.*

Post-Diphtheritic Esophageal Stenosis. (*Speiseröhrenstenosen nach Diphtherie.*) *Horst Frey, Göttingen.* Beih. Zsch. f. Hals-Nasen u. Ohrenh. 1: 500-505, Heft 11, Nov. 1949.

Only 15 cases of post-diphtheritic esophageal stenosis have been reported in the literature. A case is reported in detail in a girl of 12 years of age, and a table is presented with data relating to the 16 cases reported to date. In this patient, the stenosis developed following an attack of pharyngeal diphtheria, the first esophageal symptoms appearing three weeks after onset of the disease, before the child had fully recovered. All other possible causes of the stenosis were excluded. During the typical latent period between onset of diphtheria and onset of the esophageal stenosis, certain bridge symptoms were observed, including slight dysphagia and vomiting of blood mucus. The coughing up or vomiting of membranes is usually taken as an indication of esophageal involvement, but membranes may also be expelled through the gastro-intestinal tract, and membranes ejected from the mouth may come from the respiratory tract or stomach. The latter is not infrequently involved in diphtheria without involvement of the esophagus. In the present case, a granulation sepsis called for esophagoscopy without which the real condition would not have been discovered. The stenoses described in the literature were all cicatricial except in one instance. Possibly all cases pass through this stage. The extent of the granulations and of the necrotic inflammation will determine the time required to reach the stage of cicatricial stenosis. In the present case, the course of the disease could be followed esophagoscopically. The stenosis does not usually develop at the site of the natural esophageal constriction. In the present case it was located at the level of the second physiologic constriction, beginning 19 cm. from the upper dental margin and extending to midway between the bifurcation and the cardia to 24 cm. from the upper dental margin.

Stenosis may be so severe as to interfere with ingestion of fluids. Following the institution of a gastric fistula the patient improved probably owing to immobilization of the esophagus. There may be a coincident spasm of the esophageal musculature of primary or secondary type. The granulating esophageal mucosa may also lead to spasm exacerbating the stenosis. In granulation stenoses dilatation must be done with extreme caution, in order to avoid perforation and mediastinitis.

Various surgical measures have been resorted to in an attempt to relieve this condition, including gastrotomy, esophagotomy, gastric fistula and antethoracic plastic operation on the esophagus. Cases of

esophageal stenosis following scarlet fever have also been reported. Postdiphtheritic esophageal stenosis appears to be less frequent than formerly, possibly owing to earlier application of serotherapy. 1 table.

Abdominal Typhoid Simulated by Sphenoid Osteomyelitis Due to a Nasal Foreign Body. (*Abdominaltyphus vortäuschende Keilbeinosteomyelitis infolge Nasenfremdkörpers.*) *Horst Schumann.* HNO Beih. Zschr. Hals- Nasen- u. Ohrenh. 1: 518–20, Heft 11, Nov. 1949.

Nasal foreign bodies in adults are not so common or so easily diagnosed as in children. In some cases, such a foreign body remaining in situ since childhood, may give rise to manifold symptoms and numerous diagnostic and therapeutic problems. A case is reported in detail of a woman of 35 years, who presented symptoms suggesting typhoid fever with headache, remittent fever, roseolar eruption on the abdominal skin, enlarged spleen, and bradycardia. Herpes labialis was the only symptom apparently not related to this disease. Temporary relief followed administration of eleudron (total dose 35 Gm.) and the herpes healed. However, shortly thereafter, vomiting and headache recurred and the site of the exit of the right trigeminus became sensitive to pressure, suggesting a possible post-typhoid neuritis. The possibility of an attack of grippe was also considered. Finally consultation with neurologists as to the cause of the persistent headache, divulged left abducens paralysis of unknown etiology. The latter retrogressed after a few weeks, but was followed by renewed headaches, fever and progressive cervical rigidity. Since meningitis was suspected, the patient was re-admitted for examination. Treatment with salicylic shock and intravenous atophanyl was administered, but the cervical pain persisted. The pain subsided for a time following painful coughing up of mucus and blood.

The patient's breath was extremely malodorous and she seemed to have trouble in speaking and swallowing. The herpes was severe again and the right pupil was narrower than the left.

Finally otorhinolaryngological examination revealed greenish yellow pus oozing from the nasopharynx. There was marked pain on pressure in the region of the third cervical vertebra and lateral roentgenographic exposure revealed a large air image in front of the second and third cervical vertebrae, which were themselves normal. Suspecting an epipharyngeal abscess, the right external carotid artery was ligated to permit drainage at the base of the skull. During this intervention, the patient ejected quantities of malodorous pus from the mouth, but none escaped through the incision. Since the possibility of a gangrenous nasopharyngeal diphtheria had to be considered, the patient was given antitoxin. After the operation the dysphagia and fever

subsided and the wound on the neck healed nicely. The violent occipital headache persisted, however, with marked psychic repercussions. Finally on the suspicion of possible disease of the sphenoid sinus another examination was undertaken. This time an encrusted mother-of-pearl button was found in the right nostril in an adhesion between the septum and inferior turbinate. The incrustation indicated that the button had been retained for many years, but the patient had no memory or knowledge of its entry. She did remember having difficulty in breathing through the right nostril and suffering from severe headaches.

Following removal of the foreign body, the headaches persisted and she lost vision in the right eye. The diagnosis was retrobulbar neuritis with left abducens paresis suggesting sphenoid sinus disease. A transseptal incision of the sinus was finally done with exenteration of diseased tissue. A few days later the headache had subsided.

Idiopathic Haemotympanum. *D. Ranger, London, England.* J. Lar. & Otol. Lond. 63: 672–81, Nov. 1949.

Two cases are reported in which there was a blue appearance of the tympanic membrane as a result of altered blood in the middle-ear cleft which had occurred for no apparent reason. The condition is uncommon and only seven other cases appear to have been reported; all the cases have been unilateral.

In the two cases reported here the condition failed to clear as a result of repeated aspirations, and x-rays in one case showed clouding of the mastoid cells. Accordingly mastoidectomy was performed and both cases have remained clear for many months after this procedure. The histological sections of the mastoid were characterized by the presence of cholesterol crystals and the possible source of these is discussed.

The etiology of the condition remains obscure, but three reasons are given for the opinion that eustachian obstruction is not the essential cause of the condition: 1) Eustachian obstruction is very common but this condition is rare; 2) Eustachian obstruction has not been present in all cases; 3) The most definite cause of hyperaemia *ex vacuo* must occur in acute otitic barotrauma, but in that condition haemorrhage into the middle ear cavity occurs in only a small proportion of cases and is always accompanied by other signs such as congestion of the drum and interstitial haemorrhages.

In treatment, mastoidectomy appears to be indicated if the condition fails to clear as a result of repeated aspirations of the middle ear and particularly if x-rays show mastoid involvement. 10 references. 4 figures.

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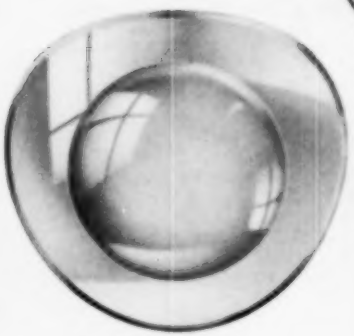

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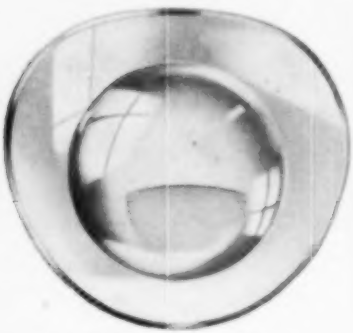

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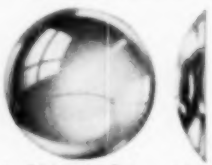

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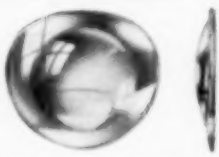

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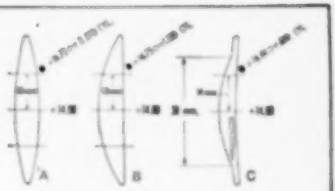

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